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TUBERCULOUS CAVITATION AND TRANSPLEURAL DECOMPRESSION

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IN THEIR paper on giant tuberculous cavities of the lung, Coryllos and Ornstein[1] stated that, in their judgment, very little progress had been made in the knowledge of tuberculous cavities during the last ten years. However, in their paper, which included an exhaustive review of the literature as well as the views existing prior to this time, Coryllos and Ornstein made a noteworthy contribution to the study of cavitation in the lung and immeasurably stimulated further progress in this field. In pointing out that bronchial disease plays a decisive role in the genesis and sometimes in the disappearance of tuberculous cavities, these authors contributed a concept of fundamental importance. (See diagram, page 19 of the above quoted paper.)

Immediately prior to the publication of this paper, Shipman[2] presented a paper before the Clinical Section of the Thirty-Third Annual Meeting of the National Tuberculosis Association in Milwaukee, Wisconsin, emphasizing the bronchial factor in cavitation. It was pointed out that certain patients react poorly to compression, that their cavities enlarge rather than disappear with such therapy, and that the chief factor in such cases was the presence of disease in the bronchus or bronchi draining the cavities involved. It was pointed out that "col-

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lapse therapy has so occupied the attention of clinicians during the past two decades, particularly as far as cavity closure is concerned, that new points of view have been long overdue. There undoubtedly exists a group of cavities in which closure may be influenced by factors other than pulmonary collapse, and in which the old rule of thumb (pneumothorax, phrenicectomy, thoracoplasty) is not applicable. This group constitutes today one of the most baffling clinical problems confronting the physician, a problem which urgently demands solution. . . . *An open mind in this matter is probably as important as an open bronchus.*"

Eloesser[3] had already emphasized the great importance of bronchial disease and stenosis in pulmonary tuberculosis and more recently, in a study of cavitation and bronchial disease in living patients, had come to the conclusion that a valve mechanism was important in the production of cavities.[4] This was in marked contrast to the view of Coryllos, who believed that cavities always close by virtue of a completely blocked bronchus, and who devoted his efforts, through a series of ingenious methods, to establishing permanent bronchial closure thus bringing about cavitary healing.

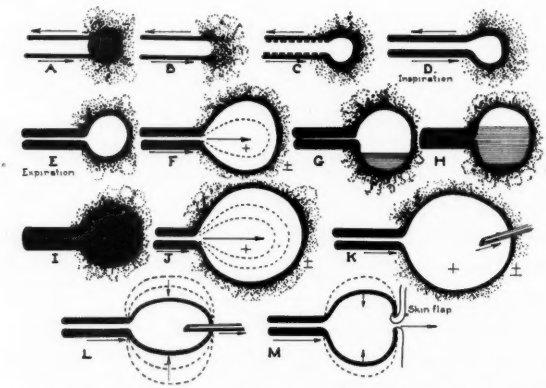

Fig. 1.—*A*, Small caseous focus; bronchus normal. *B*, Caseous focus liquefied and extruded; bronchus still normal; no cavity results. *C*, Caseous focus in process of liquefaction and contents being extruded; air and caseous material enter and leave cavity freely. *D*, Bronchial wall exhibits allergic swelling; air enters freely; egress of air and caseous material difficult. *E* and *F*, Cavity rapidly develops; bronchus partly open on inspiration and closed on expiration. *G* and *H*, Fluid accumulates in cavity; air begins to absorb; complete bronchial block. *I*, Air absorbed; fluid inspissates; "Assman" focus results. *J*, Bronchus allergic; air continues to "blow up" cavity with marked positive pressure. *K* and *L*, Tube inserted into cavity producing neutral pressure; cavity shrinks and may disappear. *M*, Skin flap and permanent opening made into cavity, which then disappears.

We believe, however, that the western school has come to accept two of Coryllos' views:

1. Certain cavities form as a result of partial bronchial occlusion, and this occlusion acts as a one-way valve blowing up the structures distal to the block.

2. Cavities *may* heal by total occlusion of the draining bronchus.

In the latter event, however, it is our belief that the so-called Assman focus frequently results and that healing may be incomplete because total block leads to retention of the secretions as well as the air, and,

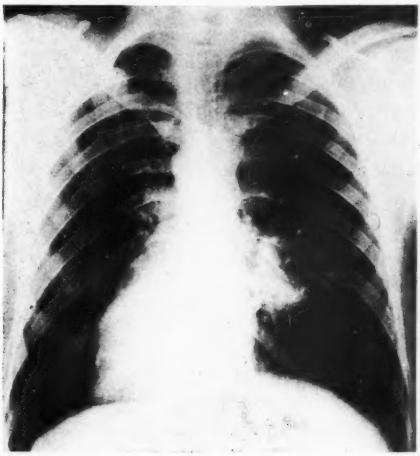

Fig. 2.—A case of complete situs inversus, showing inspissated mass in left lower lobe (which has the characteristics of the right lower lobe).

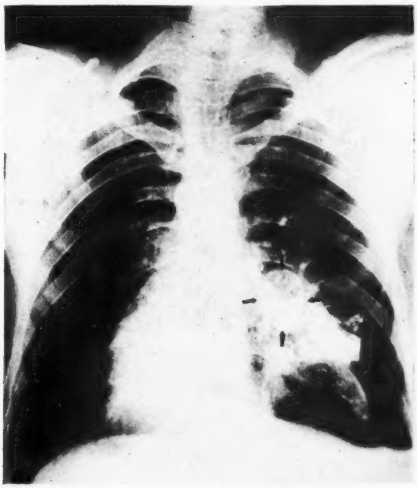

Fig. 3.—Nov. 24, 1939. Same case as shown in Fig. 2, with inspissated mass expectorated, hollow, the lower lobe partially filled by lipiodol and partially compressed by a pneumothorax.

when the latter is absorbed, there remains a more or less spherical solid body which appears to absorb much more slowly than the air, or to remain for an indefinite period in status quo, or to expel its contents and again appear as a cavity in the same location (Fig. 1). We have seen

several examples of such cases and have found that a fluid level develops, slowly rises as the air is absorbed, and apparently remains for years as a more or less inspissated mass (Figs. 2 and 3).

PRESENT CONCEPT

Our present concept of pulmonary cavitation may be said to rest upon the primary consideration that the lung acts as an elastic air-containing body tending to contract in whole or in part toward the hilum; but, if extensively diseased, it may act as a solid body, like a cheese, in which holes form through the liquefaction and discharge of the contents of caseous foci.[9]

The latter is the view that has been held by pathologists for many years. This has led to the determined use of compression therapy regardless of whether the cavities in question appeared to be compressible. The results have apparently been successful, however. Whenever the bronchus opened and neutral pressure existed in the cavity, its walls would approximate after compression therapy was adopted. Not infrequently, however, a more or less spherical or ovoid cavity surrounded by compressed lung tissue remained, in spite of all attempts to approximate the walls. This led to the development of innumerable procedures such as mobilization of the apex, removal of the costal cartilages or transverse processes, and the like, all designed to force closure of cavities which remained open regardless of relaxation of the surrounding tissues. The failures of thoracoplasty called loudly for a revision of ideas, not operative procedures.

The pathologic concept of the extrusion of caseous material in a cheeselike lung which was inelastic left much to be desired and failed to explain the formation of spherical cavities in an elastic lung. An additional concept, based upon the mechanics involved, seemed essential. A glance at Fig. 1 will clarify our present concept of cavitation in an elastic lung. It will be seen at once that two factors are usually necessary:

1. A disturbance of the integrity of the lung structure (caseous focus).

2. Bronchial stenosis.

As a rule neither factor alone will produce cavitation. Both are necessary.

The disturbance of lung integrity needs little comment. However, if a liquefying focus is present alone, it seems likely that its contents will be discharged; the surrounding tissues will shrink owing to the elastic structure of the lung; and no cavity will result, as shown at *A* and *B* (Fig. 1). It must be true, however, that inflammatory involvement of the bronchus draining such an area frequently produces a very efficient valve mechanism. We believe that allergic inflammation may be the basis for the production of these changes in the bronchial wall.

The evidence for this hypothesis is as follows:

1. A study of pathologic material reveals definite changes in the bronchial mucosa capable of producing stenosis (Figs. 4 and 5).

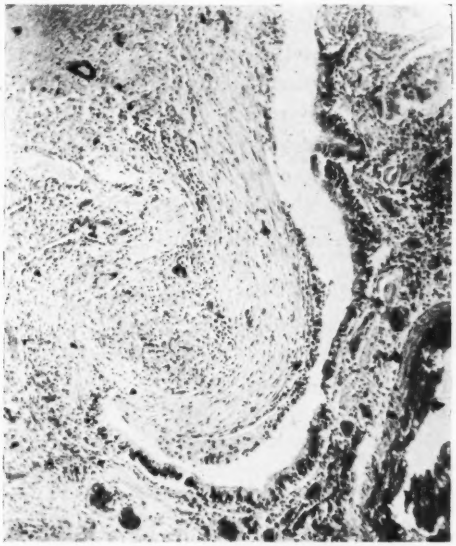

Fig. 4.—Small bronchus showing narrowed lumen, but intact epithelium. Early lesion in submucosa. Probably not completely reversible.

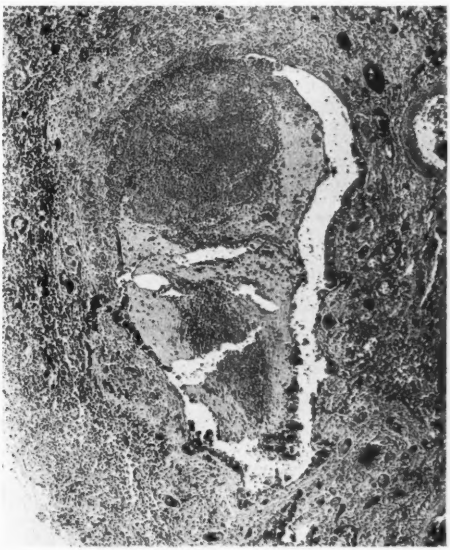

Fig. 5.—Small bronchus with intact epithelium, edema of wall, and debris within lumen. Reaction at least partly reversible.

2. Bronchoscopic observations indicate a sequence of events compatible with the known clinical picture of allergy in tuberculosis, such as skin allergy. But in this instance the allergy is of the bronchial wall. A patient suddenly develops localized wheezing. Through the bronchoscope the mucosa of the bronchus leading to the involved side is seen to be intensely red and edematous without localized ulceration or necrosis. The suddenness of the onset and the violence of the reaction, together with the bronchoscopic appearance, leave no other interpretation except that of an acute allergic tuberculous bronchitis. If this phenomenon occurs in bronchoscopically visible bronchi, it is entirely reasonable to assume that it occurs distal to the visible bronchi.

Other mechanisms causing partial bronchial block have been described. Kjergaard[5] described the mechanism of block operative in the formation of emphysematous blebs. Salkin, Cadden, and McIndoe[6] have described a number of mechanisms producing or aiding in the production of the blocked pulmonary cavity. It seems likely that all of these factors may play a role in the development and maintenance of cavitation, but that allergic swelling plays a not inconsiderable role may be assumed from the fact that the condition appears to be reversible.

CONDITION REVERSIBLE

It has already been mentioned that often, when a lung has been compressed by pneumothorax and a cavity has failed to disappear in spite of what appeared to be adequate collapse, disappearance will sometimes occur when the lung has been allowed to re-expand. It is necessary to assume that some change has taken place during the period of compression which alters the mechanics of the bronchus-cavity relationship on re-expansion. We believe that this change is a subsidence of the allergic reaction in the bronchus which allows free ingress and egress of air and secretions on expansion. The disappearance of the cavity in these cases is usually prompt and occurs as speedily as though the cavity had been opened by some other method, such as by needle, tube, or open operation. We have observed fourteen such cases, and Steele and his associates[7] have reported others.

Monaldi[8] and others have reported a large series of cavities treated by the introduction of catheters with subsequent aspiration by constant suction. A large percentage of these cavities remained closed after the catheter had been withdrawn or extruded. Some change had taken place here in the mechanics of the cavities so treated, and it seems to us not unlikely that the mechanism may have been the same, at any rate some reversible condition existed during the time of the production of these cavities, which was changed by aspiration.

In one of our cases an open operation was done, using a skin flap, and this was followed by permanent closure of the cavity. Here, again, the

condition leading to cavity formation appeared to be reversible. At this same time, we began to study the pressure relationships in cavities and to experiment with their closure. The experiences with our first case are therefore of interest.

W. W., a 30-year-old white male dentist, developed pleurisy with effusion on the right side in February, 1937. The effusion was tapped several times, and the patient returned to work. In August, 1938, however, he began to cough, and consulted Dr. Philip H. Pierson and Dr. Wm. L. Rogers of San Francisco, who referred him to the Tuberculosis Division of the Ross General Hospital after inducing a pneumothorax on the right side. The upper lobe was adherent, however, and after a few weeks the pneumothorax was abandoned. At this time the sputum was positive, and the x-ray revealed infiltration and cavitation of the right upper lobe. On entry, September 7, the patient weighed 134 pounds. On December 15 he weighed 136 pounds; the cavity was still open; and it was decided to do a first stage thoracoplasty. Accordingly, the upper three ribs on the right side were removed, and on Jan. 27, 1939, four more were sectioned. In neither instance was the periosteum destroyed (Fig. 6).

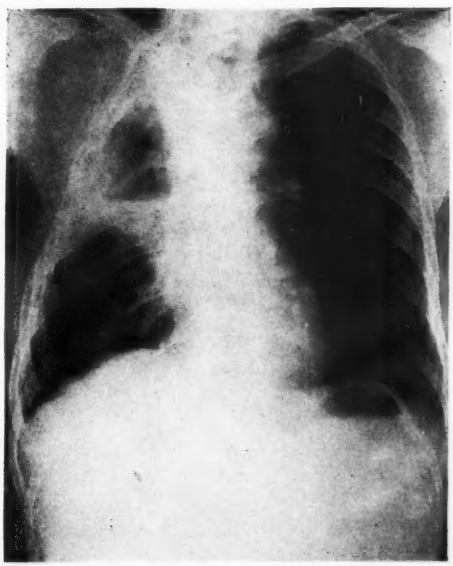

Fig. 6.—Case 1. W. W. After two-stage thoracoplasty large cavity persists in right upper lobe. Intracavitary pressure +10 +20 cm. of water.

Convalescence from these procedures was entirely uneventful; but the large cavity in the right upper lobe enlarged rather than diminished in size, and the chest wall did not fall in but remained in its original position, almost as though no ribs had been removed. This suggested to us that the underlying upper lobe, and especially the giant cavity in it, were under markedly increased pressure or tension, due to a bronchial valve factor as described above. A 16 gauge needle was therefore inserted into the cavity, and pressure readings taken (+20 +12 cm. of water). In order to decompress the cavity, the needle was allowed to remain in it. Within

twenty-four hours the cavity was no longer visible. After a few days the needle was removed, whereupon the cavity resumed its previous size.

From this time until June 5, 1939, a 16 gauge needle was allowed to remain in the cavity, which seemed to shrink until it was no longer visible. On several occasions the needle was removed, after which, in a few hours, the cavity would again appear to be as large as ever. Each time the needle was reinserted, the intracavitary pressure would be approximately +20 +10 cm. of water. The valve mechanism seemed to be operative constantly.

On June 5 the needle was withdrawn and replaced by a "T" catheter. This was not well tolerated, however; the patient's temperature remained 101° to 102° F. during this period. Accordingly, the tube was removed on July 24. The cavity, which had not been visible during the catheter period, ballooned out to its original size within five days (Fig. 6). Both the sputum and cavity drainage were constantly positive for tubercle bacilli.

Since simply exposing this cavity to atmospheric pressure resulted in its disappearance, it was decided to open the cavity permanently. Accordingly, on July 31, 1939, a U-shaped incision was made in the axilla; a section of the fourth rib was removed; and the thickened pleura overlying the cavity was removed with the cautery. The right upper lobe was seen to be a large cavity lined by dirty gray necrotic material, and one large bronchial fistula was visible. The skin flap was sutured to the upper margin of the opening into the cavity with two chromic gut sutures, in order to protect the axilla. The cavity was then packed with iodoform gauze.

Fig. 7.—Case 1. W. W. April 25, 1940. Nine months after decompression by Eloesser flap into cavity. No sputum. Flap healed. Cavity closed.

On the following two days, the patient's temperature rose to 105° F., although he stated he felt quite well. Since it was believed that this was due to the iodoform pack, the latter was removed and vaseline gauze was substituted. The patient's temperature promptly fell to 100° F. The cavity rapidly became smaller until within

a few weeks nothing was left except a sinus tract leading to the bronchial opening. This closed in February, 1940. A right temporary phrenic operation was done on March 22, 1940. By April 22, the patient's weight had risen to 149 pounds; his temperature was normal; and there was no sputum or drainage from the wound (Fig. 7).

ROLE OF BRONCHI

In approximately 100 cases of tuberculosis autopsied at the San Francisco City and County Hospital during 1939, careful examination of all bronchi was made. Special consideration was paid to bronchi opening into cavities. Every cavity examined showed open bronchi numbering anywhere from one to six. In no instance were closed bronchi found.

If allergy plays a dominant role in the early pathogenesis of cavities, autopsy findings will usually be of little value. Practically all cases with high allergy do not die early in the formation of their cavities unless some unusual pathologic entity is present. Rarely we find lesions in small bronchi which suggest an allergic reaction. By allergic reaction we mean a marked edema and congestion of the wall of the bronchus, together with a few cells and an intact bronchial epithelium. This type of lesion may well be reversible. The usual type of bronchial lesion seen at autopsy, with ulceration, fibrosis, absorption of cartilage, and destruction of muscle, is irreversible.

At times lesions exist in small bronchi and bronchioles which, of course, cannot be seen by the bronchoscope, but these lesions are relatively uncommon at autopsy.

Figs. 4 and 5 are histologic sections showing bronchi frequently encountered at autopsy.

Fig. 4 shows a narrowing of the bronchial lumen with an intact epithelium. In the submucosa is an early lesion of the bronchus which shows early connective tissue with a few chronic inflammatory cells and no evidence of edema. Acid-fast stains on the section were negative. Serial section showed that ulceration had occurred in one small area with rupture of a small caseous focus into this bronchiolar lumen. It is doubtful if this reaction is reversible.

Fig. 5 shows a small bronchus with an intact bronchial epithelium and marked edema of its wall, cells of the chronic inflammatory type, and some debris within the lumen. Acid-fast stain was negative. This reaction in the bronchiole may be partially reversible.

These slides and our autopsy findings cannot prove that allergy is a dominant factor in the early pathogenesis of cavities. Tuberculin tests are indicated in all patients in whom this mechanism is suspected, but skin sensitivity does not necessarily parallel visceral sensitivity. Probably the only definite means of proving the role of allergy would be by controlled animal experimentation.

By means of continuous kymographic recordings of intracavitary pulmonary pressures in patients undergoing transpleural decompressive therapy, we were able to show that certain physiologic changes occur in these cavities. We are able to show that changes in the intracavitary pressures are only infrequently related to the actual breathing of the cavity—that is, the actual flowing of air into the cavity with inspiration (Fig. 8). In tension cavities this phenomenon seems to occur rarely

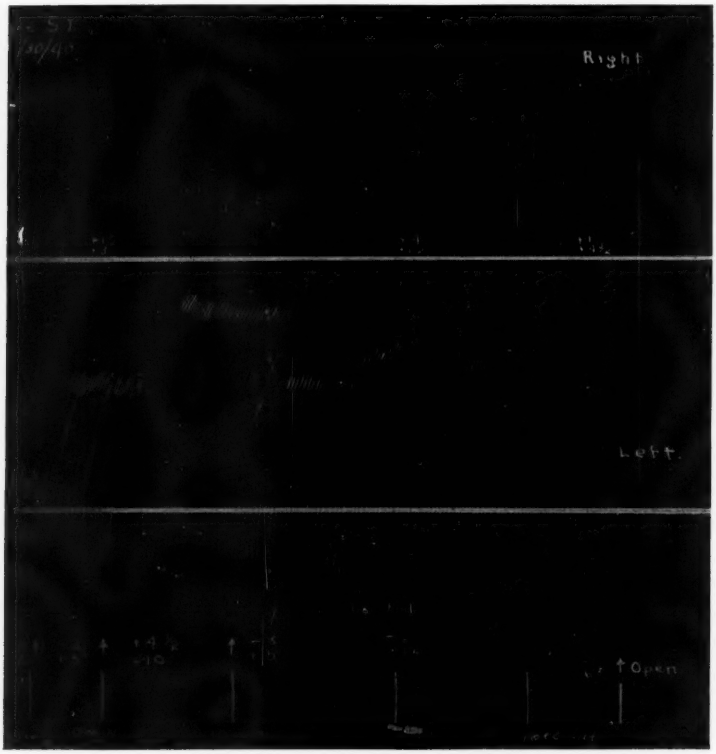

Fig. 8.—Case S. T. Bilateral simultaneous kymographic tracings of intracavitary pressure changes. Bronchus open in upper tracing (right). Bronchus obstructed in lower tracing (left). Both cavities negative for tubercle bacilli. Tube removed on left four weeks later. Note the marked tension of cavity on the right, positive in both phases of respiration.

during the daily life and actions of such a patient. Calculating the amount of air actually leaking into such a cavity during twenty-four hours would give an answer (usually under 10 c.c.) in a cavity whose volume was approximately twice this amount. Furthermore, the flow of air from the bronchus into the cavity does not occur bit by bit with each inspiration or expiration, but occurs only at very infrequent periods over the course of a few respirations (Fig. 8). The condition under

which this leakage of air can be induced artificially occurs when we induce a negative pressure in the cavity.

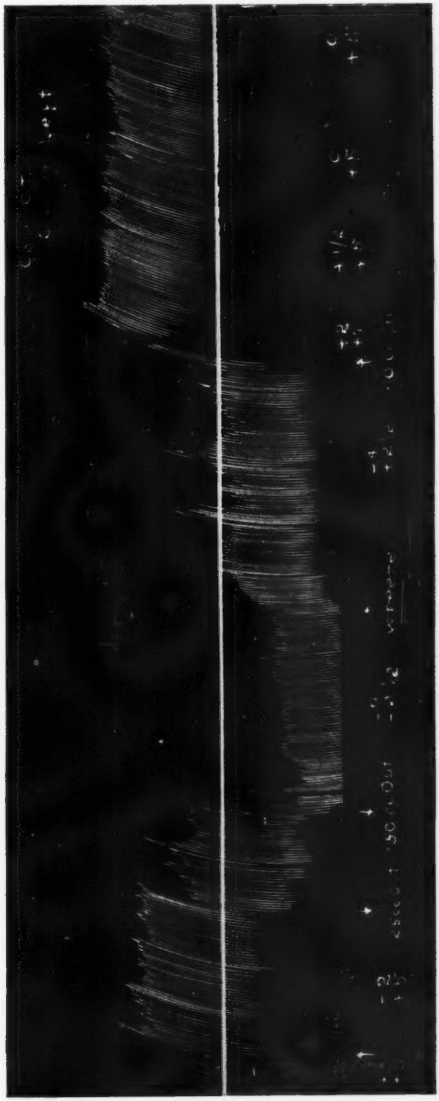

Fig. 9.—Case C. T. Kymographic tracing of intracavitary pressures showing that obstructive effect is from bronchus to cavity and not from cavity to bronchus. Leakage of air from bronchus to cavity at "wheeze," occurred with artificially induced negative pressures. A few minutes later when the intracavitary pressures were artificially raised at "50 c.c. in," no leakage of air from cavity to bronchus was demonstrated.

That the bronchus-cavity relationship at most times is valvular, with the valve prohibiting the passage of air from cavity to bronchus, has been proved in a number of experiments (Fig. 9). Air was introduced transpleurally into the cavity so that the pressures in the cavity were

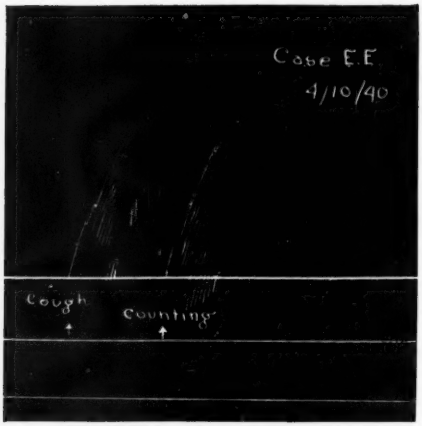

Fig. 10.—Bronchus open; cavitary pressures raised by cough, and counting pressures descend in steps.

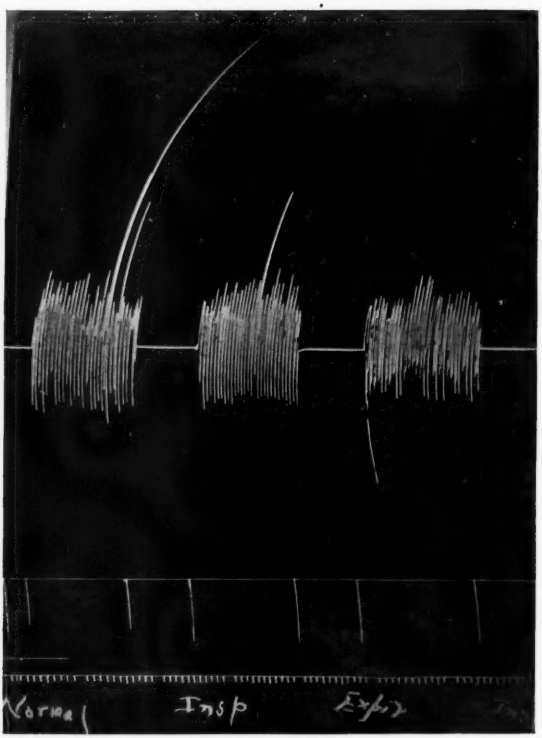

Fig. 11.—Case L. F. Showing failure of attempts to maintain increased or decreased pressure by introducing or sucking out air. The bronchi were patent without an efficient valve mechanism.

elevated to approximately +5 +20 cm. of water. In no instance did a decrease of these pressures occur during the times of our experiments. The efficiency of the valve was 100 per cent—or a complete stop-valve action occurred—so that no air could flow out of the cavities through the bronchus at the pressures used over a period of five to thirty minutes, totaling several hours.

In certain cavities, after a period of transpleural decompression, the cavity-bronchus relation showed evidence of change. Thus, in at least three instances which had a stop-valve action early in their course of decompression, an opening had occurred which allowed the pressures to fall when they were artificially raised by the injection of air, and which allowed free ingress of air when a negative pressure was induced (Fig. 10).

A type of large cavity was also observed which had a cavity-bronchus relationship incapable of showing valvular effects at the times of our experiments; no deviations were ever observed on several attempts during a period of ten months (Fig. 11). In all the experiments the artificially induced negative pressures or positive pressures were never maintained. An unobstructed nonvalvular bronchus-cavity relationship was present and was stable and unchanging. This was not a tension cavity, its pressures being bronchial –0 +1½.

Briefly summarizing the findings, we may say that kymographic tracings of intracavitary pressures show that:

1. In tension cavities respiratory fluctuations of pressure are caused by the pressures transmitted through the walls of the cavity from their surrounding lung.

2. In tension cavities very little actual flow of air in and out of the cavities takes place through the bronchus.

3. In tension cavities the valve formation is usually 100 per cent efficient in the cavity-to-bronchus direction. In these cavities the pressures may be positive in both phases of respiration.

4. The valvular efficiency of a cavity-bronchus relationship has been demonstrated to change during the course of transpleural decompression therapy.

5. Nontension cavities may have pressures simulating the normal bronchial pressures.

6. Nontension cavities have no demonstrable valve mechanism.

7. Nontension cavities have a flow of air in and out of the cavity with each respiration.

In all, we have attempted transpleural decompression on twenty cavities in eighteen patients (two bilateral cases) (Figs. 12 and 13). In seven instances the treatment was not completed, either because of

failure of cooperation on the part of the patient, an apparent aggravation of the existing tuberculosis, or serious complications such as tension pneumothorax (two cases).

Fig. 12.—S. T. Simultaneous bilateral cavimography. On the left bronchial obstruction has occurred. On the right the bronchus is still patent. Both sides have no tubercle bacilli in discharge. Tube removed on right four weeks later.

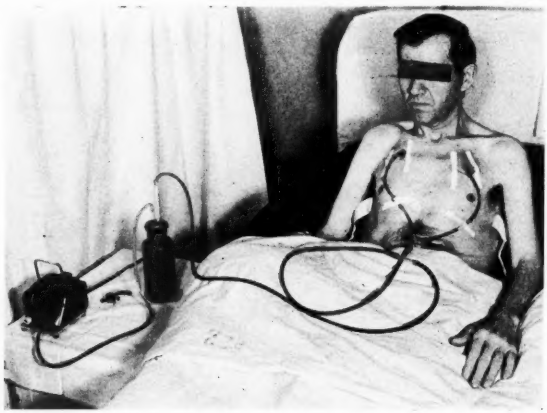

Fig. 13.—Case S. T. Apparatus for transpleural decompression. Simultaneous bilateral motor suction using mechanical pump.

There were three deaths in this series. Both patients who developed tension pneumothorax and empyema died. In both of these cases, preliminary pneumothorax attempts were not adequate; in one instance only one attempt was made. This led to the dictum that pneumothorax attempts should be made in at least five different locations. A third death was caused by tuberculous pericarditis, apparently unrelated to the treatment.

Two cavities are apparently permanently closed. One of these was treated by transpleural decompression followed by skin flap, the other by transpleural decompression alone. Five additional cavities show secretions negative for tubercle bacilli, but the catheters are still in place. In one case, the cavity is still open by x-ray, but the catheter has been removed and the sputum and secretions have been negative for tubercle bacilli for the past six months.

Two patients had thoracoplasties after transpleural decompression was discontinued. In both of these cases, the cavities are still open. Four patients had thoracoplasties prior to transpleural decompression. One of these patients is apparently cured. In one, transpleural decompression is being continued; the cavity is very small; and the secretions have been negative for tubercle bacilli for more than a month. The third cavity is smaller, but the secretions are still positive for tubercle bacilli. The fourth is open, and the sputum is positive.

COMMENT

A theory of bronchus-cavity relationship has been proposed, based upon transpleural decompression studies, and the behavior of certain cavities under collapse therapy and after this therapy has been abandoned. According to this concept, the production and maintenance of certain cavities is dependent upon the presence of bronchi-cavity valves which allow the passage of air in one direction only, i.e., toward the cavity. From observations of the disappearance of certain cavities following the abandonment of pneumothorax, we have come to believe that an allergic reaction in the bronchus may be highly important. This phase deserves further study. If correct, the subsidence of the allergic reaction during transpleural decompression might explain the behavior of cavities undergoing this treatment.

Our observations on twenty cavities undergoing transpleural decompression seem to disprove the theory that atmospheric oxygen must be prevented from reaching the cavity in order that its tuberculous infection should die. Cavities seem to disappear when the bronchi are wide open and when they are closed.

In every instance, the cavities chosen in this series were thought to be unsuitable, for the usual compression measures or collapse therapy had already been unsuccessfully attempted. The failures of standard compression therapy would seem to demand a new approach based upon the physiology of cavitation. Transpleural decompression, at least in part, seems to answer this demand.

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(See discussion on page 522.)

THE CHOICE OF PROCEDURE IN THE TREATMENT OF TUBERCULOUS CAVITIES

Considerations Regarding Intracavitary Pressure Based on Clinical Observations and Experiments on Cadavers

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THREE fundamentally different therapeutic methods occupy the attention of those interested in the surgical treatment of tuberculous pulmonary cavities. The first of these are methods of collapse; the second are methods of compression; and the third are methods of aspiration or drainage, open or closed. More direct aggression by wide unroofing of cavities, or by resection in the form of lobectomy or pneumonectomy, will not be considered here.

The three widely used methods—collapse, compression, and aspiration—postulate such fundamentally different ideas of cavity formation and cavity healing that it has seemed useful to try to inquire why each method has succeeded in closing certain cavities and failed to close others, to see whether it may be possible to distinguish various types of cavities and to select for each cavity that procedure most likely to obliterate it. Many cavities have been closed by all of these methods, yet the choice has been more or less haphazard since precise indications for a choice of procedure have not been formulated.

Collapse treatment in the narrower sense presupposes that a lung which is freed from pull of the chest wall will collapse by virtue of its elasticity, retract toward the hilum, remain immobile, and no longer take part in respiratory movement. It presupposes that cavities, like the rest of the lung, are kept distended by this pull and that measures relieving them of it will either cause them or allow them to collapse. Thus an artificial pneumothorax is induced; if the cavity fails to collapse, it is thought that failure is usually due to adhesions which still keep it under tension. The adhesions are severed. If extensive adhesions preclude a pneumothorax, thoracoplasty is done; and, if a cavity still persists, more extensive thoracoplasty, additional rib resection, scapular resection, resection of transverse processes are resorted to—possibly in the hope of freeing the recalcitrant lung from persistent pull of structures holding it open, possibly in the hope of converting what was begun as a collapse operation into a compression operation. Certain facts, however,

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cast doubts on the universal value of collapse. There are, for instance, cavities that remain open and protrude in the form of a bubble from a lung which is elsewhere well collapsed by pneumothorax; others remain open in spite of thorough adhesion cutting, both closed and open; others, after apical thoracoplasty, retain their original size and are pushed downward in the chest instead of collapsing; while others miraculously close after an inadequate attempt at rib resection at the hands of some tyro. This strange behavior of some cavities, both their resistance to apparently adequate collapse measures and their disappearance after apparently inadequate ones, troubled many observers, and in 1937 in a paper before this society I endeavored to explain it by the assumption that some cavities, at least, were kept actively inflated by a valve mechanism of the communicating bronchus. If this assumption is correct, then releasing the lung from the pull of the chest wall should not collapse these cavities, they not being pulled open from the outside, but pushed open from the inside, as it were.

Perhaps one factor in the collapse type of operation has not received due attention. Singer and Adams, and Coryllos and his coworkers have repeatedly emphasized the effect of atelectasis on the healing of tuberculosis. Atelectasis or lack of it may account for the inadequacy of some collapsing procedures and the cures resulting after others which were apparently inadequate. Atelectasis results wherever circulation of blood in the lung is maintained and circulation of air abolished. Anything that will make the lung stop moving will cause the air in it to be absorbed, provided blood continues to circulate through it. Whether healing is favored by immobility or airlessness, or both, and whether immobility and airlessness are the crucial factors, or factors at all, remain to be seen; it is reasonable, however, to suppose that tuberculosis will come to a standstill more readily in a quiet organ than in one that is constantly moving.

If efficacy of collapse depends upon release of tension and production of atelectasis, it may be reasonable to suppose that methods of treatment based upon these factors may cause cavities to close when they are held distended by external tension; if, however, there are cavities that are ballooned up from within, then this variety should resist measures depending on release of external tension for their effect, unless indeed the collapse is accompanied by changes in the anatomy of the communicating bronchus, which either open it, so as to release the pressure within the cavity, or perhaps seal it off entirely and allow the air in the cavity to be absorbed. Collapse operations, therefore, should be more efficacious in the treatment of open communicating cavities, i.e., those in which the manometer swings equally to both plus and minus sides of the manometric scale when they are needled, from which methylene blue and lipiodol are promptly expectorated and which do not appreci-

ably change when air is aspirated from them or injected into them. They should be used with hesitancy, if at all, for cavities which show a constant high positive pressure when needled and from which methylene blue and lipiodol are not promptly expectorated. If aspiration of large amounts of air closes a cavity and injection balloons it up, it should be subjected to thoracoplasty, if at all, immediately following closure by aspiration, before it has a chance to become reinflated (Rogers).

Compression methods for closing cavities, methods intentionally directed toward squeezing a cavity down, pressure pneumothorax, pressure with gauze and paraffin packs have fallen into disuse, if we except extrapleural pneumothorax, in which considerable pressure seems to be necessary to counteract the retractability of the pneumothorax wall. The idea of closing an abscess by squeezing it down is repugnant to surgical practice. The danger of perforating a cavity by exerting pressure on it is too great to sanction pressure methods. While it is not impossible that complete immobilization and complete atelectasis by pressure on the surrounding lung may perhaps favor absorption of air and closure of a cavity, patients upon whom pressure with packs has been tried have not had their cavities closed in our hands.

Aspiration and suction drainage presuppose a cavity in which pressure is higher than that in the surrounding lung *most of the time,* so that it is actively ballooned out from within. Observations reported to this Association in 1937[1] seem to show that these conditions prevail part of the time in many cavities. If a cavity when needled contains air under positive pressure, if the manometer shows little or no swing, or in swinging remains on the positive side, if methylene blue and lipiodol are retained in the cavity, if aspiration closes the cavity or diminishes its size, suction seems indicated and a method that would tend to keep the cavity disinflated seems reasonable. However, Coryllos[2] at the next annual meeting criticized the method sharply, perhaps not unreasonably, stating that by suction I had opened the previously closed bronchial connection and had converted closed cavities into open ones, to the detriment of the patient and asked how I could hope to obtain a collapse by suction. Since then observations have multiplied, and the question of cavity formation and cavity treatment has occupied the attention of numerous workers. Monaldi and a large staff at the Forlanini Institute in Rome have worked out a method of suction drainage in much detail; his method has been followed and is being followed by numerous workers in many countries. Dr. Kupka, of Olive View, is reporting his experiences at this meeting.

With so active and widespread an interest a progress report may be justifiable, even before definite conclusions have been reached.

In the hope of further elucidating intracavitary pressure eight cadavers were studied, five with tuberculous cavities, one with marked bronchial stenosis, and two with normal lungs. The chest was opened, and the lungs were connected with a

large bellows of 3.5 L. capacity by a wide tube fastened into the trachea. This tube had a T-tube interposed leading to a water manometer. The ordinary bellows stroke projected about 1.2 to 1.5 L. A second manometer was connected to a needle with which the lungs were punctured. Working the bellows produced uncommonly instructive movements of the lungs, simulating shallow or deep inspiration, cough, etc. A swing of 6 to 12 cm. of water pressure was produced. Sudden quick pressure simulating a cough registered from +10 to 14 and higher. These cadaveric respiratory movements were thought to be especially illuminating, for the lung lay free and unhampered by the chest wall; whatever variation occurred was due to changes in the lung itself; the intracavitary and intrapulmonary pressures were uninfluenced by extrinsic factors. Incidentally, when a stethoscope was applied directly to the cadaveric lung, various qualities of breath sounds and various kinds of râles could be heard with such clarity that it seemed as though the method might prove valuable in teaching physical diagnosis to students, especially since the pathology underlying the abnormal sounds could be demonstrated forthwith.

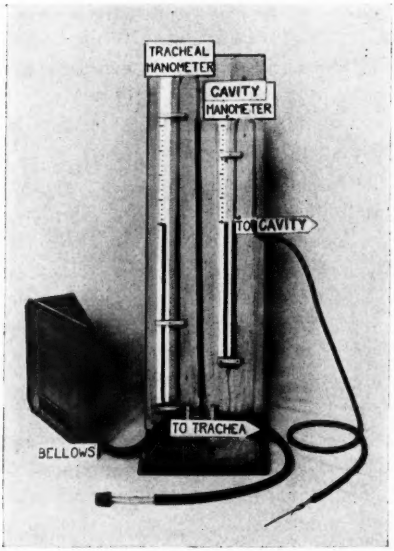


Fig. 1.—Apparatus for simulating respiratory movement, needling cavities, and measuring intratracheal and intracavitary pressure in cadavers.

CADAVER 1.—The lungs of the first cadaver were not needled, as antemortem films were not at hand and no cavities could be palpated. The lag of the affected right lung, however, was shown very clearly when breathing was simulated with the bellows.

CADAVER 2.—A white stevedore, aged 28 years, had pulmonary tuberculosis diagnosed two years previously and died April 2, 1940. X-ray examination on Feb. 20, 1940, showed extensive bilateral apical tuberculosis with bilateral cavitation. At autopsy the left upper lobe was found to contain multiple thin-walled cavities. In the right upper lobe at the level of the second interspace was a multilocular cavity 4 to 5 cm. in diameter, with a fibrous wall 8 mm. thick. The cavity was lined with a dirty gray exudate. It communicated with a small branch of the upper lobe bronchus, which could be easily probed. The middle lobe was moderately firm; the lower lobe was moderately wet.

When the lungs were inflated with the bellows, all lobes moved freely except the right upper one, which moved little if at all. The right upper lobe cavity was needled, and about 5 c.c. of pus were aspirated. The lung was again inflated. Only after considerable pressure was built up in the lung was there any movement of the

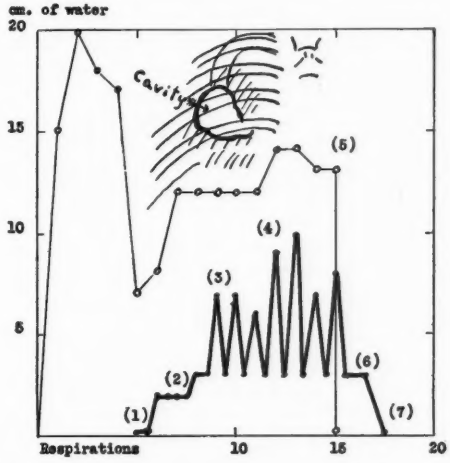


Fig. 2.—Cadaver 2. Only inflation bellows-pressure is noted; on releasing the bellows, the pressure in all cases fell to zero. (*1*) Cavity needled. (*2*) Cavity partially blocked (ball-valve effect). (*3*) Cavity communicates with bronchus, but retains a positive pressure even at the end of the expiratory phase. (*4*) Increased force of breathing. (*5*) Respirations stopped. (*6*) Pressure in cavity remains at +3. (*7*) Cavity manometer disconnected from needle; manometer drops to 0. Thin upper line indicates intratracheal pressure; heavy lower line, intracavitary pressure. Insert: Large irregular cavity of right apex six weeks antemortem.

cavity manometer. However, when a pressure of 12 cm. of water was reached in the tracheal manometer, the cavity manometer ran to 7 cm. and swung from +7 to +3. When the bellows pressure was released, the tracheal pressure dropped to zero, but the cavity pressure remained at +3. It was concluded that considerable pressure was required within the bronchi before direct connection could be established with the cavity. However, when this pressure was reached, the cavity communicated freely with the bronchus, its pressure fluctuating with each respiratory movement. When pressure in the trachea was released, pressure in the cavity remained elevated, indicating that its bronchus was blocked. When the manometer was detached from the needle, the reading fell to zero, again indicating that there must have been positive pressure within the cavity. The observation was recorded with a moving picture camera. The film was shown at the Cleveland meeting of the American Association for Thoracic Surgery. A graph (Fig. 2) was made from the camera record by Dr. Philip Westdahl. The cavity was partially blocked and would have been suitable for thoracoplasty, aspiration, or a combination of the two procedures.

CADAVER 3.—This observation was discarded since the final position of the needle was doubtful.

CADAVER 4.—A female clerk, aged 39 years, had had a cough of two months' standing and a right apical cavity. She died of intestinal tuberculosis with perforation. There were several cavities in the middle of the left upper lobe about 2 cm. in diameter. There were a few fresh tubercles in the left lower lobe. The right

pleural cavity was obliterated by adhesions. There were multiple tortuous, connecting cavities occupying the right upper lobe and most of the right middle lobe. The walls of the right upper lobe cavities were only 2 mm. thick in many places and consisted mostly of pleura. The cavities connected with each other and with a large patent branch of the right upper major bronchus. Section of this bronchus showed extensive tuberculous ulceration and caseation.

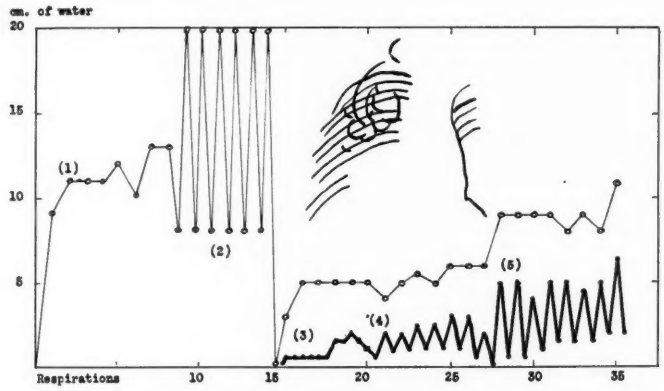


Fig. 3.—Cadaver 4. (*1*) Ordinary respirations. (*2*) Series of coughs. (*3*) Cavity needled but remains partially blocked. (*4*) Cavity communicates freely with bronchus, respiration shallow. A little air is entrapped in the cavity; pressure does not quite reach base line. (*5*) Depth of respiration increased; a little more air is entrapped in the cavity. Thin upper line indicates intratracheal pressure; heavy lower line, intracavitary pressure. Insert: Large irregular cavities of right apex (postmortem roentgenogram).

The breastplate was removed but the lungs were left in situ in the thoracic cavity, a cannula was inserted into the trachea and attached to a bellows by means of which artificial breathing was conducted. The following findings were noted: (1) The left lung expanded more than the right. (2) The right lung was moderately contracted. (3) On quiet respiration the large cavity in the right upper lobe at first remained collapsed. (4) After three to four respirations the cavity ballooned out while the rest of the lung remained quiet. (5) When breathing was stopped, the cavity remained ballooned out. (6) A 19 gauge needle was inserted into the cavity and connected to a manometer. (7) The variations in the cavity manometer followed those in the main tracheal manometer, but the height of the fluctuation was a few centimeters less in the cavity manometer. (8) After a few artificial coughs the pressure in the cavity rose, but always returned to a positive pressure at +1. (9) One cubic centimeter of India ink was injected into the cavity. (10) On cutting down upon the needle, it was found to be in the center of the cavity and the India ink had spread throughout all of the compartments. The procedure was recorded with a camera; the film was shown at the Cleveland meeting. Fig. 3 was plotted by Dr. Philip Westdahl.

CADAVER 5.—A waiter, aged 27 years, had large bilateral apical cavities and died on Aug. 6, 1940. The right apical cavity had been needled on June 22, 1940. A 22 gauge needle, too fine for accurate measurements, was used. The manometer readings were as follows: When the cavity was first entered, no manometer variations occurred; the patient counted to seven, still no variation; he coughed, the pressure rose to +7 and receded slowly in about one minute to a swing of + and −1 (communicating bronchus blocked). He was told to sniff; no variation occurred.

He coughed a second time; the pressure rose to +5 and fell promptly to +1 (communicating bronchus open). He sniffed a second time; the pressure fell to −2 but promptly receded to +1 (communicating bronchus open). An attempt at aspirating air from the cavity with a 50-c.c. syringe was unsuccessful, probably because of the fine needle. Lipiodol and methylene blue were injected. Methylene blue appeared in the sputum three hours later. The communicating bronchus was partly blocked during the first cough and sniff and was open following the second cough and sniff. The patient had no reaction from the needling.

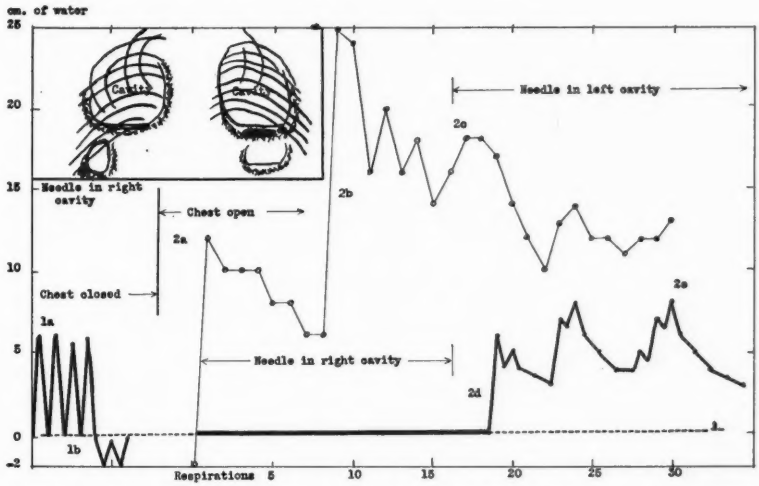


Fig. 4.—Cadaver 5. Note: Only peaks of intratracheal pressure are noted; pressure drops to zero each time bellows are released. (*1*) Chest closed. (*1a*) Needle introduced into cavity at right apex through closed chest. Manual compression of chest wall. Cavity pressure rises when chest is compressed and drops to zero when compression is released. (*1b*) Chest closed. Chest expanded by traction on cord introduced through lower intracostal spaces. Cavity pressure falls with traction and rises to zero when traction is released. (*2*) Chest open. (*2a*) Bellows attached to trachea. Needle in cavity at right apex. Cavity apparently blocked. No fluctuations in cavity manometer. (*2b*) Series of artificial coughs fails to open cavity. (*2c*) Cavity at left apex needled. Cavity blocked and not affected by coughs. (*2d*) Coughs open cavity which then communicates with bronchus by valve mechanism, producing constantly positive cavity pressure. (*2e*) Respirations stopped. Cavity pressure slowly drops and remains positive. Air entrapped in cavity. Fine upper line indicates intratracheal pressure; heavy lower line, intracavitary pressure. Insert: Roentgenogram four weeks antemortem. Large cavities of both apices. Lipiodol and fluid levels after needling left cavity.

The left cavity had been needled on July 11, 1940. Again a 22 gauge needle, too fine a one, was used. On first introducing the needle no manometric swing occurred. The patient was told to sniff; the pressure fell to −3 and remained at this level (communicating bronchus blocked). He counted to seven, and no variation in pressure occurred. The sniff was repeated; the pressure dropped to −3, −4; the sniff was repeated again, and the pressure was −5. This evidently opened the communicating bronchus, for the pressure suddenly rose to +2. Now, when the patient counted to seven, the pressure rose to +3, immediately returning to neutral. Suction with a large syringe produced no change in the size of the cavity. Lipiodol and methylene blue were injected at 1:15 P.M.; the dye had appeared in the suptum by 6:00 P.M. The maneuvers were followed by a rise in temperature to 100° F., which lasted one day.

At autopsy the left apical cavity measured 8 by 8 by 7 cm.; its wall was 2 mm. thick at the thinnest portion. Anteriorly and laterally it was fibrous, the lower

portion was formed by compressed tissue (?) lung (?). At its base were two small bronchi 2 mm. in diameter, one entering directly into the base of the cavity, the other running obliquely through its wall. Another cavity 3.5 by 2.5 by 2 cm. communicated with the first one by two openings 0.5 cm. in diameter.

The right apex was completely occupied by a large cavity 8 by 5.5 by 8 cm. Its wall was 0.5 cm. thick at the thinnest portion. Below its medial portion was a small bronchus which arched through the lung parenchyma and entered the anterior wall.

Before the autopsy was begun, a needle was introduced into the right apical cavity through the intact chest wall. Rhythmical compression of the lower chest wall produced a free swing of intracavitary pressure, the manometer returning to zero when pressure was released. Two stout cords were passed around the lower ribs with a needle, and rhythmical traction was made on the chest; the manometer fell each time the cords were pulled, returning to zero when traction was released. However, the fall in pressure produced by traction was not so great as the rise produced by compression.

The breastplate was then removed, and the right apical cavity was again needled. Now neither quiet respiration nor violent compression of the bellows succeeded in causing fluctuation of intracavitary pressure. The cavity was apparently blocked. It would seem, therefore, that the manometric variations produced by compressing and making traction on the chest wall when the cavity was needled through the intact thorax were imparted to the air in the cavity by movements of the surrounding lung acting through the thin cavity membrane. When the chest was opened and the lung freed from restraint of the rib cage, the surrounding lung no longer influenced the cavity; the cavity was blocked and failed to respond to movements of the bellows. *This observation indicates that respiratory variations of intracavitary pressure may be imparted to a thin-walled cavity by the surrounding lung and are no proof that the communicating bronchus is open.*

With the chest open, the left apical cavity was also needled. At first the needle registered no pressure, but a few sudden puffs on the bellows simulating coughs opened it, the pressure rose to +7 and constantly remained high. Further coughing forced the pressure up; it never dropped back to zero, but retained a high level for some time after respiratory movement had ceased, falling off slowly to +2. Air was therefore entrapped in the cavity. The communicating bronchus ran for some distance through its wall, entered it on a slant, and acted as a valve, being shut off by increasing intracavitary pressure.

These observations indicate that even irregular, ragged walled cavities situated in a densely infiltrated, largely airless area of lung contain air which is under more than atmospheric pressure a considerable part of the time. This is true even under conditions of normal respiration; this pressure rises and may remain high for considerable periods after coughing or straining against a closed glottis. The observations are the more striking as they were made on cavities with shaggy, necrotic walls, which we are not accustomed to consider as typical of blocked or ballooned cavities, but rather as ones with an open bronchial communication. The bronchial communications were indeed open, but not freely so; secretion, pus, and shaggy bits of necrotic material were probably sufficient to block them partially part of the time, so that it may not be improper to assume that inflation by entrapped air plays a role in maintenance or production of these cavities also.

The lungs of two cadavers with normal lungs were needled as controls. After a short period without manometric variations the water column began to swing back and forth freely. This was found to be due to the formation of a small air space, a traumatic cavity, produced where the point of the needle had torn the lung a little during its respiratory movements. It was interesting to note that air remained entrapped even in this little cavity and that the manometer, once it began to swing, failed to reach the base line but continually registered a few centimeters of positive pressure.

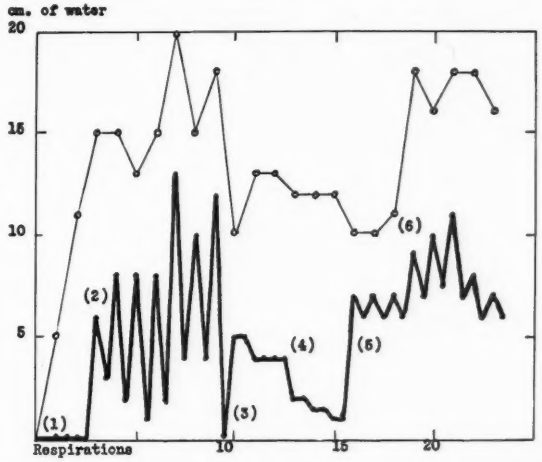


Fig. 5.—Puncture of normal lung. (*1*) Lung parenchyma needled with 14 gauge needle; no effect. (*2*) Needle withdrawn slightly; free fluctuation begins; air entrapped in traumatic cavity. Pressure does not reach zero. (*3*) The 14 gauge needle removed and an 18 gauge needle inserted into parenchyma in different location. (*4*) Needle partially obstructed. (*5*) Needle withdrawn slightly; free fluctuation begins; more air entrapped in traumatic cavity; cavity pressure constantly positive. (*6*) Depth of respiration increased. Note: Only peak of tracheal manometer fluctuations are noted; each time bellows are released, pressure drops to zero. Fine upper line indicates intratracheal pressure; heavy lower line, intrapulmonary pressure.

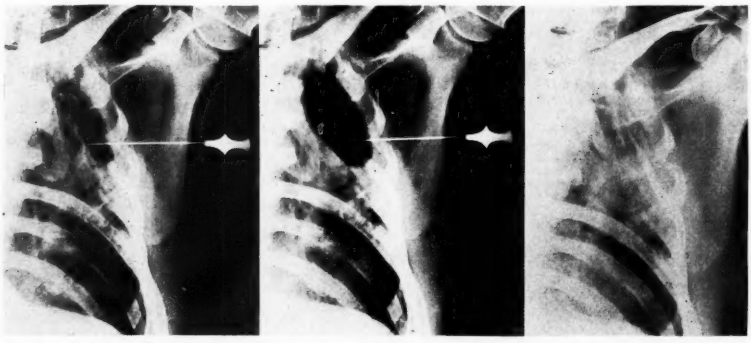
Cavities have been needled in over sixty patients, in some patients repeatedly. No untoward result, empyema, serious infection of the chest wall, hemorrhage, or collapse has been observed. The needling is done under the fluoroscope. A 19 gauge, 2.5- to 3-inch needle is used, attached to a 2 c.c. Luer syringe. After the cavity is reached, a two-way stopcock is attached to the needle; one end is connected with a manometer, the other, to the syringe. Pressure is noted on first entering the cavity. The patient is told to count to ten; pressure is noted, and a note made as to whether the pressure returns to a neutral swing promptly. If it remains positive, it is assumed that the cavity is blocked, at any rate at the time at which the reading was taken. The patient is told to sniff with the nostrils held closed. Pressure and return to neutral are again noted. Five to ten cubic centimeters of air are alternately injected and aspirated while the walls of the cavity are

watched under the fluoroscope to see whether they dilate or contract. Two cubic centimeters of aqueous methylene blue and 1 c.c. of lipiodol are injected. The lipiodol is watched under the screen to see whether it enters a bronchus, and the patient is told to note whether methylene blue appears in the sputum and, if so, when. Finally, at Dr. Rogers' suggestion, as much air as possible is aspirated from the cavity. It has been found possible by this simple means to close a number of cavities entirely. After the cavity has been closed, readings are again taken. Throughout the procedure a number of small spot films are made. Finally the needle is withdrawn, and several standard exposures are made with the patient erect.

Rogers' method of immediate closure of cavities by aspiration has in several instances produced most dramatic results. He has followed aspiration by thoracoplasty in order to prevent re-expansion and in two cases has injected 0.6 c.c. of 10 to 20 per cent silver nitrate with the idea of sealing the communicating bronchus. This injection, however, failed to accomplish the desired result.

CASE REPORTS

CASE 1.—A. R. E., a patient of the Weimar Joint Sanatorium, was seen by Dr. Rogers through the kindness of Dr. Thoren and Dr. Briner. A large cavity had reopened in the left apex in spite of two thoracoplasties with removal of seven ribs. The cavity was needled on March 17, 1940. The cavity pressure readings were as follows: Initial pressure, + ½ to + 3. Straining against closed glottis: + 12. Sucking against closed glottis, – 8. Counting to about ten: + ½ to + 3½, pressure rising

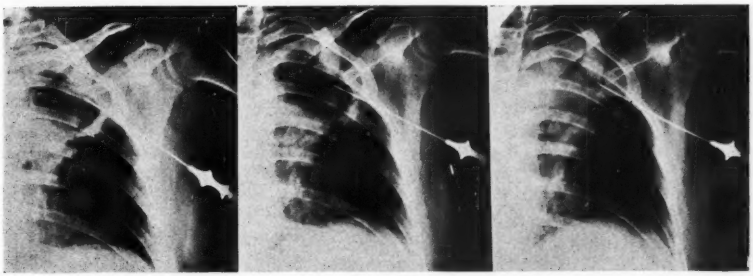


A. *B.* *C.*

Fig. 6.—Case 1. *A*, 20 c.c. of air aspirated, cavity contracted. *B*, 40 c.c. of air injected, cavity double size. *C*, 40 c.c. of air aspirated; needle withdrawn, and cavity no longer visible.

with each word. Twenty cubic centimeters of air were aspirated; the cavity contracted greatly (Fig. 6*A*). Twenty cubic centimeters of air were replaced, and 20 c.c. more were injected (total 40 c.c.). The cavity was double the former size (Fig. 6*B*). Forty cubic centimeters of air were aspirated, and the needle was removed; the cavity was no longer visible and was apparently closed (Fig. 6*C*). This manipulation was followed by a rise of temperature to 99.8° F., lasting two days. Three days later the cavity had reopened.

CASE 2.—E. S., a patient of Dr. Thoren and Dr. Briner, had a left apical cavity, unclosed after pneumothorax and phrenic interruption. Dr. Rogers needled the cavity on March 17, 1940. Initial pressure: +2 +5½; straining, +10 +15; counting, +5½; sucking, −10. Twenty-five cubic centimeters of air were injected; the cavity expanded. As much air as possible was aspirated; the cavity closed (Fig. 7*A*, *B*, *C*). The cavity reopened by the third day. The temperature rose to 100.2° F. on the second day and returned to normal on the sixth day.



A. *B.* *C.*

Fig. 7.—Case 2. *A*, Cavity needled. *B*, 25 c.c. of air injected; cavity larger. *C*, All air aspirated; cavity no longer visible.

CASE 3.—Mrs. B. C., a patient at the Tulare-Kings Sanatorium, was referred by the kindness of Dr. Winn. A right apical thoracoplasty had failed to close an apical cavity. On April 25, 1940, Dr. Rogers needled the cavity with a 19 gauge needle. Initial reading: +2, +3; after one minute return to 0 with barely perceptible variations. Counting to ten: +4, immediate return to neutral. Straining: +4, immediate return to neutral. Sucking: −6, immediate return to neutral. Thirty cubic centimeters of air were aspirated; the pressure was −5, −7; the pressure slowly receded, remaining stationary after about five minutes at a level of −2, −3. After this, counting and straining were repeated. Straining reopened the cavity which attained only one-third its original size. Suction of 30 c.c. of air again closed the cavity (manometer −6). Methylene blue injected into the cavity did not appear in the sputum up to twenty-four hours. The cavity remained closed twenty hours later. Thoracoplasty was done on April 26, the fourth, fifth, and sixth ribs and the tip of the scapula being resected. The patient was put to bed on her right side with instructions to hold her nose closed and to sniff every hour. A film taken May 10, 1940, failed to demonstrate a cavity; the whole right lung was airless. The patient was rolled over onto her left side; hereby the lower lung was reaerated, but the cavity remained closed.

CASE 4.—Mrs. W. F., patient of Dr. Nay of San Luis Obispo, had a large cavity of the left apex. Dr. Rogers needled the cavity on April 6, 1940. Initial readings: +1, +2½; counting ten: +1, +5, immediate return to neutral; straining: +9½; suction: −8. Aspiration of 65 c.c. of air apparently closed the cavity. 0.65 cubic centimeter of 10 per cent silver nitrate was injected in the hope of closing a bronchial communication. In the hope of maintaining the closure, thoracoplasty with resection of the first to fourth ribs was immediately performed by Dr. Rogers. The patient was seen again on May 18, 1940, after an uneventful convalescence. The cavity had reopened. Aspiration of 15 c.c. of air sufficed to close it a second time, whereas 65 c.c. were aspirated before. 0.5 cubic centimeter of 10 per cent silver nitrate was injected into the cavity, and closure was effected by resecting three more ribs (fifth, sixth, and seventh).

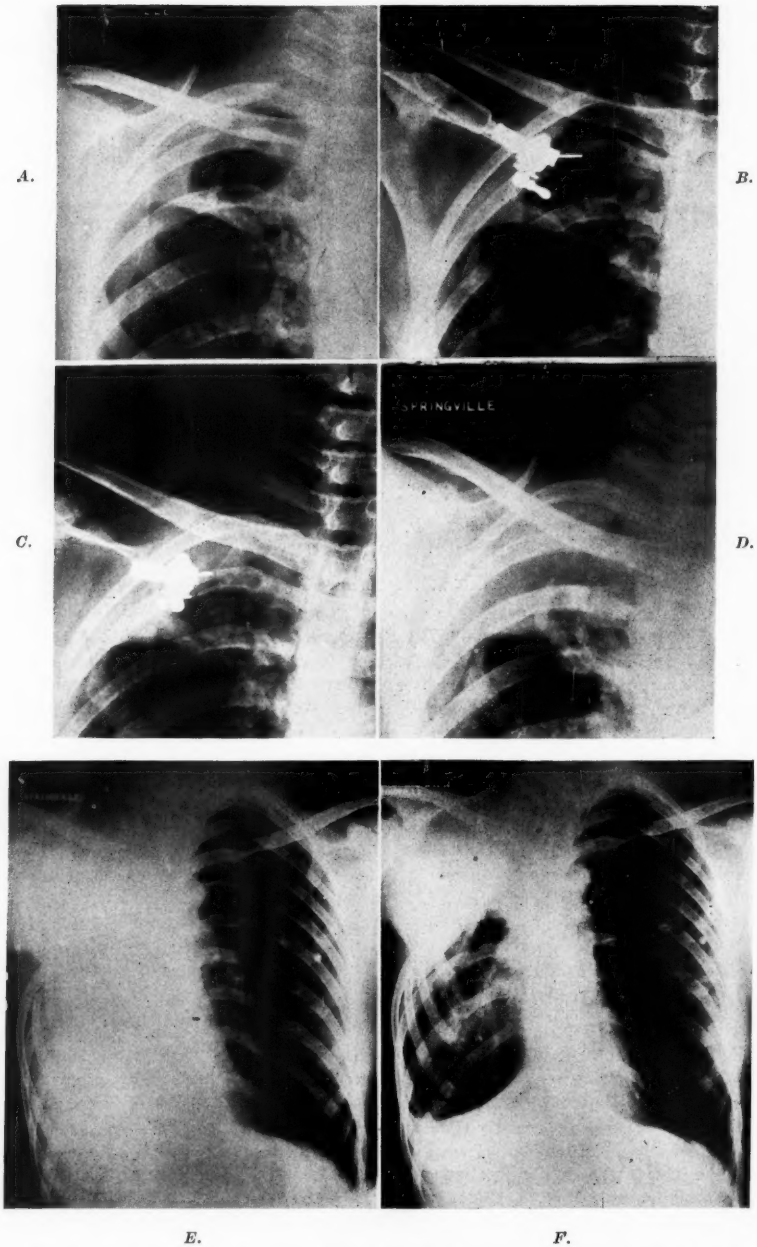


Fig. 8.—Case 3. *A*, Right apical cavity (April 9, 1940). *B*, Cavity needled. *C*, 30 c.c. of air aspirated; cavity no longer visible. *D*, Needle withdrawn; cavity not visible. *E*, Two weeks after second stage thoracoplasty. Patient had lain on right side and had been told to sniff every half hour. Right lung airless. *F*, Patient put on left side and made to cough; right base unfolds, but cavity does not reopen.

CASE 5.—Mrs. R. B., patient of Dr. Schultze at the San Mateo Community Hospital, had a right apical cavity, uncollapsed by thoracoplasty. On March 25, 1940, under the fluoroscope a needle was introduced into the cavity. One had the feeling that it traversed thick pleura. Pressure in the cavity fluctuated from − to +1 on

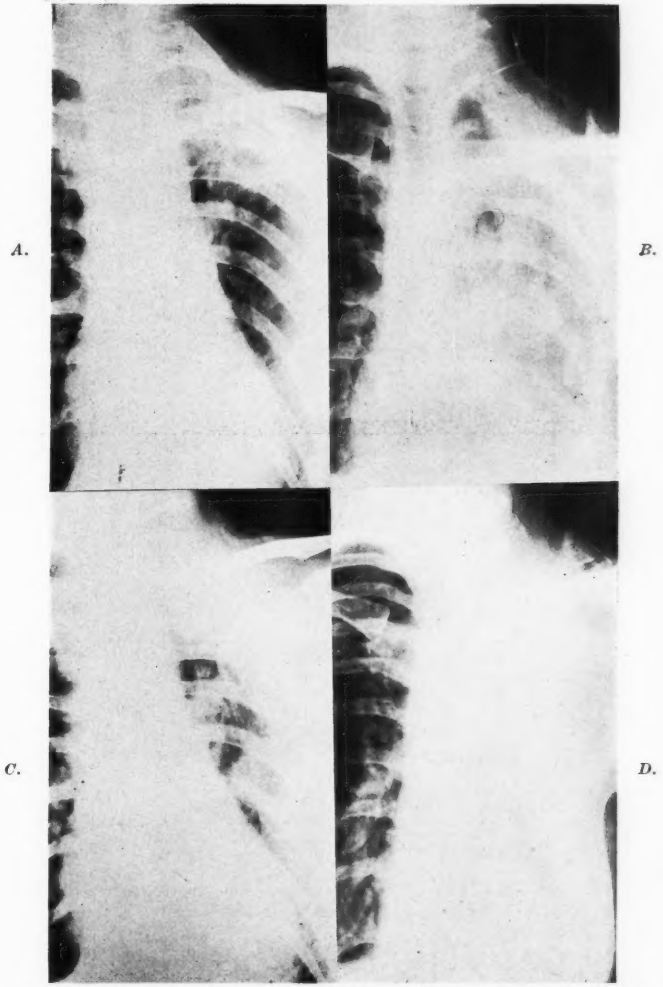


Fig. 9.—Case 4. *A*, Left apical cavity before thoracoplasty (April 16, 1940). *B*, Twelve days after aspiration of cavity and resection upper four ribs; cavity has reopened. *C*, Reaspiration of cavity; resection of fifth, sixth, and seventh ribs; cavity closed. *D*, Six weeks later; cavity remains closed; left lung atelectatic.

quiet breathing; sniffing caused a pressure of −6, which returned promptly to +1, −1½. Counting to 7 made a positive pressure of +5 which returned promptly to +1, −2½. Aspiration of 17 c.c. of air and 1 or 2 c.c. of fluid after air was aspirated produced a pressure in the cavity of −7. There was no stopcock on the needle and

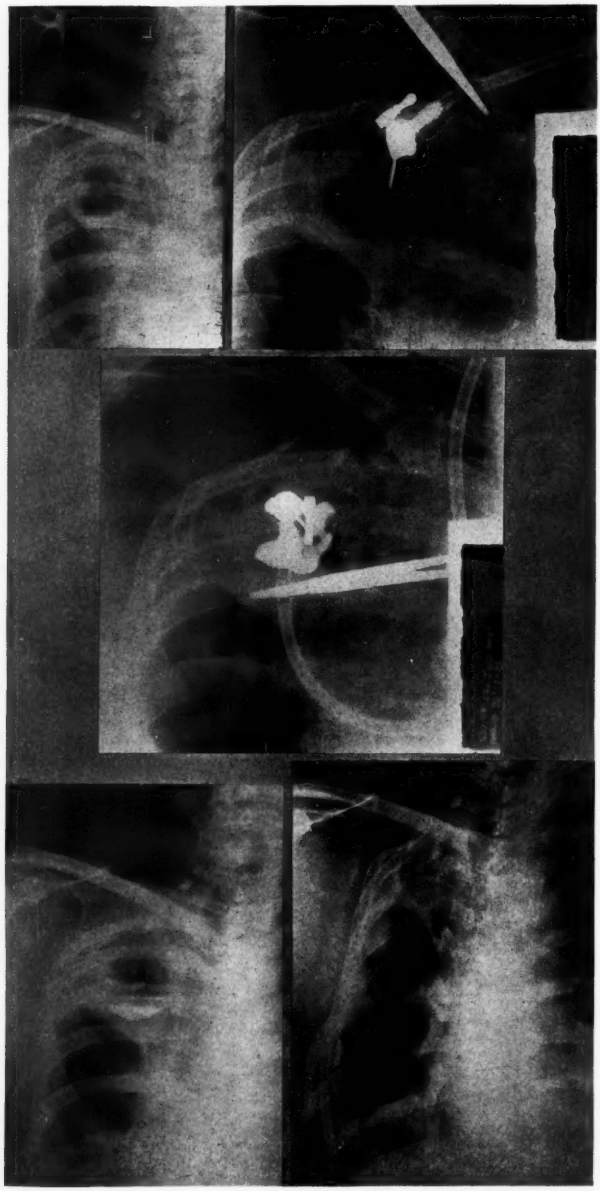


Fig. 10.—Case 5. *A*, Right apical cavity uncollapsed after thoracoplasty. *B*, Cavity needled (March 25, 1940). *C*, 20 c.c. of air withdrawn (March 25, 1940). Cavity no longer visible. *D*, Cavity reopened (March 29, 1940). Cavity contains lipiodol. *E*, Nov. 6, 1940, 3.5 months after second stage thoracoplasty. Cavity no longer visible.

air re-entered the cavity. It was reaspirated. Again 17 c.c. were withdrawn with a little fluid following the air. A manometer was connected to the needle. Readings in the cavity were – 3 to 0. On sniffing there was a pressure of – 10, returning promptly to 0; on counting to 7, a pressure + 3, returning promptly to – 3, + 0. Aspiration of the cavity therefore succeeded in producing a certain degree of negative pressure, although the pressure readings would indicate a free bronchial communication. A two-way stopcock was now applied to the needle, and 20 c.c. of air were withdrawn. The cavity seen under the screen appeared to be closed. Then 25 c.c. of air were introduced. The cavity reopened. This experiment was repeated several times. Finally methylene blue and lipiodol were introduced, neither of which the patient coughed up. Neither sniffing nor coughing produced a variation in the manometer after the two-way stopcock was applied. The needle was withdrawn, and films were taken. The cavity seemed to be closed. Lipiodol was seen in it. Films two days later again demonstrated the cavity, which had reopened. On July 29, 1940, the cavity was again needled and closed by aspiration. This maneuver was immediately followed by resection of four more ribs. The patient convalesced smoothly. The cavity remains closed, and expectoration is greatly diminished. The scanty sputum occasionally contains tubercle bacilli; at other times none are found.

Two patients in whom suction drainage had failed to close a cavity were operated upon following a suggestion of Dr. Shipman by inserting a skin flap into the cavity, making a semiclosed drainage path.

CASE 6.—Mrs. P. P., aged 21 years, a patient in the San Francisco Tuberculosis Hospital, was found a year and a half before entry to have a widely disseminated, left-sided tuberculosis with a large apical cavity. She was toxic, febrile, and emaciated. Pneumothorax had failed to influence the cavity and had been allowed to recede. The cavity was needled on Oct. 28, 1939 (Fig. 11 *C, D*), and suction drainage instituted. The cavity walls moved a few millimeters when the plunger of a 10-c.c. syringe was moved back and forth. Intracavitary pressure on quiet respiration was –4, +2. Methylene blue injected into the cavity was promptly expectorated, and lipiodol ran into the communicating bronchus. The cavity was judged to be freely open. Suction drainage was followed by marked diminution of expectoration, gain in weight and defervescence, but the cavity remained apparently unchanged in size. Therefore suction was discontinued from Jan. 18, 1940, to March 13, 1940, although the catheter was allowed to remain. On April 4, 1940, a long U-shaped flap of skin, its base in the axilla, was turned up; 5 cm. of the second rib were resected; and the cavity was entered with the cautery. It lay several centimeters below the surface of the lung and was surprisingly small—about the size of an English walnut. Its lining was smooth and fibrous, and several small bronchi were seen entering it. The skin flap was attached to its border with two catgut sutures, and the edges of the defect in the axillary skin were brought together with sutures. By September the patient's expectoration had practically ceased, and the sputum became negative for the first time. The small amount of secretion from the axillary opening no longer contains tubercle bacilli. She was discharged to her home on Dec. 28, 1940.

CASE 7.—Mrs. D. McK., aged 50 years, a patient at the Canyon Sanatorium, was referred by the kindness of Dr. E. A. Schaper. She had had pulmonary tuberculosis for four years. There was a widely disseminated right-sided tuberculosis with a large pressure cavity in the middle of the right lung field. The patient was emaciated and toxic. Catheter drainage had diminished, but not abolished expectoration, had reduced the fever, and diminished the size of the cavity. However, since the cavity could not be closed completely by catheter suction drainage, it was decided to attempt drainage with a skin flap.

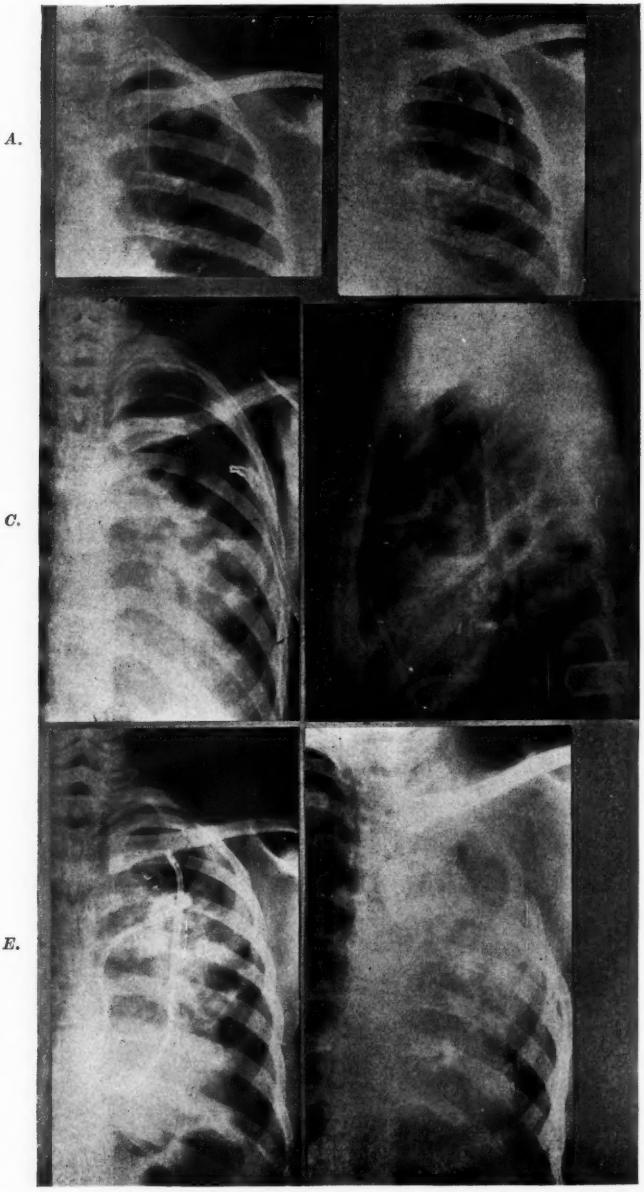
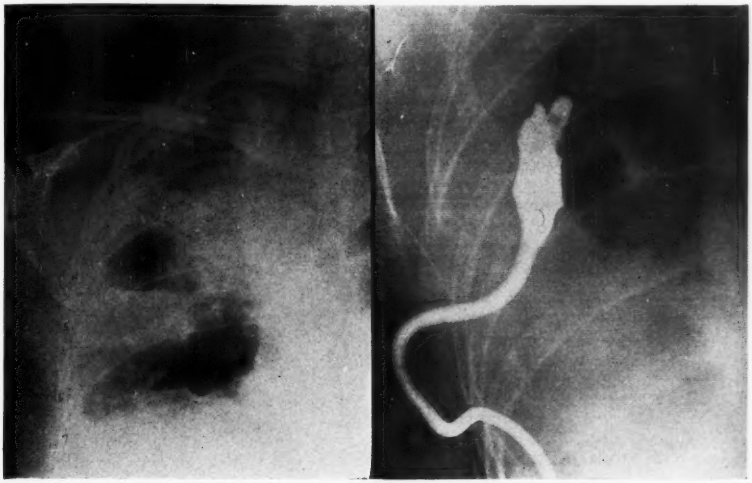
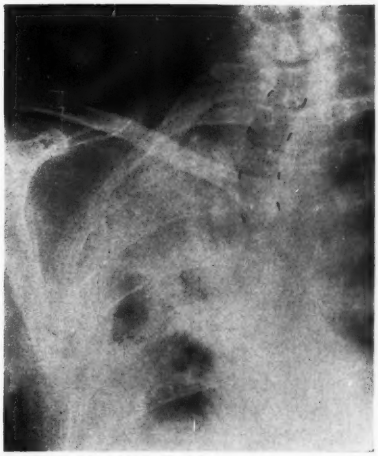


Fig. 11.—Case 6. *A*, Large left apical cavity (July 5, 1938). *B*, Cavity larger after pneumothorax (Sept. 26, 1938). *C*, Suction drainage (Oct. 28, 1939). *D*, Lateral, same. *E*, Cavity smaller (March 6, 1940). *F*, After thoracoplasty and drainage with skin flap. Cavity no longer visible (Oct. 23, 1940).

Operation.—Jan. 24, 1940. Pentothal anesthesia was used. A skin incision was made in the form of a flap with the base upward and forward and the tip downward and backward. About 1.5 inches of the right fourth rib were resected together with the periosteum. The pleura was 1 or 2 inches thick. A needle introduced through the



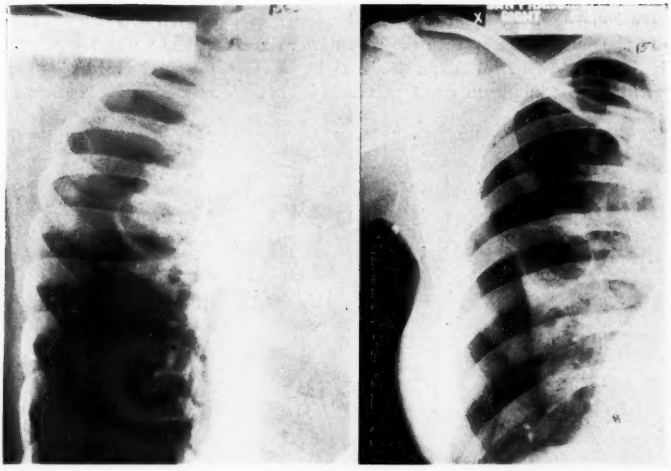
A. *B.*



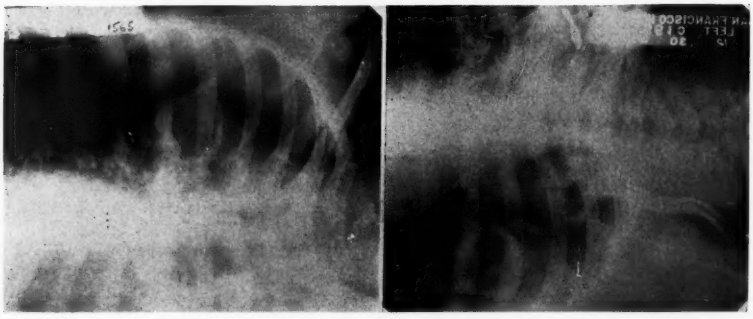
C.

Fig. 12.—Case 7. *A*, Large right soft cavity with fluid level. *B*, After drainage with flap. Lipiodol injection. *C*, Cavity no longer seen.

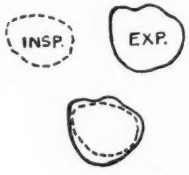
thickened pleura withdrew air from the cavity at a site more superficial than might have been supposed from the film. The needle was followed by a cautery which entered a large cavity, the wall of which was thin and friable. Suction applied to the cavity withdrew 1 or 2 ounces of thin green pus. In order to keep the patient from coughing, no attempt was made to inspect the inside of the cavity,

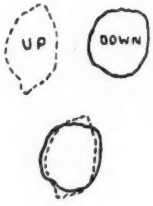


A. B.



C. D.





E.

Fig. 13.—*A*, Inspiration. Cavity elliptical. Posteroanterior projection. *B*, Expiration. Cavity more rounded. *C*, Patient recumbent on contralateral side. Cavity up. Adhesion pulls it into shape of lemon. *D*, Patient recumbent on side of cavity. Cavity down, assumes a rounded shape. Note: Cavity shown distended by pull of adhesion in *A* and *C* and released when pull is lessened (*B* and *D*). Pneumolysis indicated. *E*, Tracings of cavity.

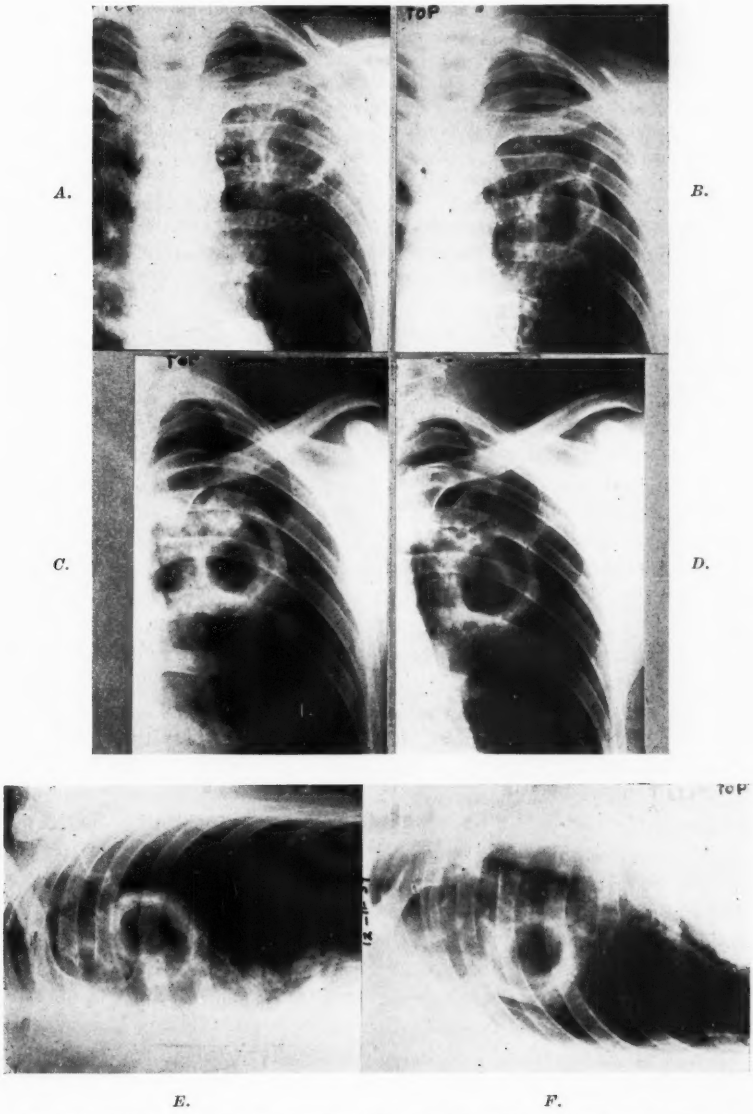


Fig. 14.—Cavity persists in pneumothorax. Pneumolysis not indicated. The adhesion does not distort the cavity, which retains its annular shape unchanged in inspiration (*C*), expiration (*D*), with the patient recumbent on the contralateral side (*E*) and on the cavity side (*F*). *A*, Left apical cavity (Sept. 12, 1939). *B*, Cavity persists in pneumothorax (Nov. 9, 1939). *C*, Inspiration (Dec. 11, 1939). *D*, Expiration. *E*, Recumbent on contralateral side. *F*, Recumbent on side of cavity.

but the tip of the flap which had been previously armed with two catgut sutures was rapidly sewn into place in the cavity and the remainder of the defect in the skin was approximated with silkworm-gut stitches.

Postoperative Note.—When the patient coughed, air was expelled from the cavity. When she lay in bed on the right side, no air was sucked back and forth. When she lay on the left side, there was a tendency for air to be sucked into the cavity. However, in any position the air was expelled easily as the patient coughed and did not easily enter on quiet respiration. A film taken a day after operation showed the cavity to be much smaller or to have been obliterated. For the first week the patient had a rather stormy course, the temperature reaching at times 39° C. The wound was dressed infrequently, and the patient kept on her right side. Hereupon her temperature dropped to 99° F. and 100° F. at night; she gained in weight; and the sputum diminished. She was discharged to her home on Feb. 26, 1940.

Blocked Cavities and Artificial Pneumothorax. Indications for Pneumolysis.—The foregoing observations apply to patients whose pleuras are obliterated. Needling, of course, is not legitimate in the presence of a free pleural space. The question of an intracavitary block, however, may also arise in patients with artificial pneumothorax. Too frequently pneumothorax is ineffectual in collapsing cavities. Indeed, as Shipman has pointed out, some cavities that persist in a large pneumothorax will vanish if the air is allowed to absorb, wholly or in part. This fact alone is evidence that it is not only adhesions that hold a cavity distended, but other forces. It is of some importance then to determine before proceeding to cut adhesions whether the adhesions are actually at fault. If the pull exerted by them is the determining factor, it would seem that changes in shape of a cavity should follow changes in the tension exerted by them and that placing the patient on one side or another, or making him inhale or exhale, should change the shape of the cavity.

Therefore before pneumolysis was done, x-rays of a number of patients with artificial pneumothorax were taken in various positions. One film was taken after expiration and another after inspiration with the patient erect; a third film was taken with the patient recumbent on the good side and a fourth with him recumbent on the affected side. Some of the films showed no change in the shape or size of the cavity. If adhesions were at fault in holding the cavity distended, one might expect that it could be pulled into an angular or irregular shape when more tension was made on the adhesions by placing the patient on his unaffected side and causing him to distend his chest. However, as several of the cavities examined retained their annular form whatever maneuver their carriers were put through, it was deduced that it was not adhesions but some other factor that kept them distended. That this is actually so was shown by a case[1] (Case 1, Figs. 2-6) in which a ballooned cavity retained its air and shape after both closed and complete open pneumolysis.

SUMMARY AND CONCLUSIONS

1. The pressure in many tuberculous cavities varies accordingly as their communicating bronchi are temporarily open or blocked.

2. It is higher than atmospheric pressure for a considerable part or most of the time, even when the communicating bronchi are fairly well open at times.

3. This pressure tends to keep cavities distended.

4. A respiratory pressure variation of intracavitary air does not prove that the communicating bronchus is open, for the surrounding lung may impart pressure variations to the cavity contents. Proof of a blocked communication is afforded, however, if intracavitary pressure remains elevated while the patient holds his glottis open and stops breathing.

5. Observations corroborating these conclusions have been made in cadavers and are reported; the methods used are described.

6. *Provided always that no free pleural space exists* needling with the purpose of differentiating open cavities, probably amenable to thoracoplasty and other collapse operations (in the strict sense of the term), and blocked or partially blocked cavities which are probably not amenable to these measures, is legitimate and useful.

7. Three cases of sudden deflation of cavities by needling and attempts at maintenance of deflation by immediate thoracoplasty as proposed by Dr. Rogers are reported.

8. Two cases of semiclosed drainage of cavities by skin flaps as proposed by Dr. Shipman are reported.

9. Thoracoplasty is not unlikely to be unsuccessful in collapsing cavities with high intracavitary pressure.

10. Whereas such a cavity may be closed temporarily by a single aspiration (Rogers),

11. or by continuous suction drainage (if it contains a considerable amount of pus),

12. in order to obliterate the cavity permanently, it is usually necessary to follow suction by secondary thoracoplasty,

13. or by drainage with a skin flap (Shipman).

14. Cavities may persist in artificial pneumothorax because of (a) fixation by adhesions or (b) intracavitary pressure.

15. Cavities held distended by adhesions should change their shape when the patient is put in various positions and when he inhales and exhales.

16. Cavities held open by increased intracavitary pressure should not show these changes.

17. Pneumolysis is not likely to close cavities which do not change their shape with changes in the patient's posture, or on inspiration and expiration.

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2. Coryllos, P. W., and Ornstein, G. G.: Giant Tuberculous Cavities of the Lung, J. THORACIC SURG. 8: 10, 1938.

DISCUSSION ON "TUBERCULOUS CAVITATION AND TRANSPLEURAL DECOMPRESSION" BY DR. HAROLD BRUNN, DR. SIDNEY SHIPMAN, DR. ALFRED GOLDMAN, AND LOREN ACKERMAN;* AND "THE CHOICE OF PROCEDURE IN THE TREATMENT OF TUBERCULOUS CAVITIES" BY DR. L. ELOESSER

DR. LOUIS R. DAVIDSON, New York.—I am going to confuse you, because I, myself am confused. It is needless for me to tell you that we have had brilliant and definitely provocative papers. Perhaps an entirely new approach to our problems—reduction and closure of cavities—lies before us. Regardless of what this treatment (intracavernous aspiration by the Monaldi technique) will mean in the light of ultimate advantage to the patient, we can say that we have an added medium for the study of pulmonary biology and physiology, particularly in its relationship to cavities.

I should like to digress and say that my brilliant predecessor at Sea View Hospital, Dr. Coryllos, would not have been, because of his profound knowledge of chest physiology—and I use this term advisedly—anything else but a guiding spirit in the direction and understanding of the possibilities of these new studies in the biology and the mechanics of the pulmonary cavity.

Now there are certain factors which I should like to take up with you. If it is true that these tension cavities, these cavities which balloon out, are entirely the result of a ball-valve mechanism, I am again perplexed. However, you have heard that both Dr. Kupka and Dr. Shipman also have been bewildered by their findings. I have found that in one cavity the operator may obtain an initial reading of −2, +6. In another case he may get a +4, +12 reading.

There is one danger, however, in the readings of those pressures. The apparatus must be intact. An airtight system must be present for the simple reason that, if air escapes or is lost from the pulmonary cavity, the manometric pressure readings are then definitely vitiated.

We have attempted a new manner of approach to this problem. Not only do we study air pressures, not only do we study the gaseous interchange, but also by means of a particular instrument which we have devised, and by the use of special telescopes, we inspect from day today, from hour to hour, the walls and contents of the pulmonary cavity. In addition, in a relatively surgical way, through this metal cannula and optical system, we have just begun to perform what I shall term ascending catheterization of the bronchi and coagulation of the draining bronchus. With these particular measures we hope, first, to study the relationship of the bronchus to the cavity and, second, to attempt to block the stem bronchus, thereby causing obliteration of this pulmonary cavity.

(Slide) This is the closest I could "steal," if you will, or copy, the original Monaldi instrument. After hearing from various men who had been with Monaldi, I was about to play with a cannula of my own design. However, about this time, I happened upon the article by Bottari and Babolini. The first slide shows a direct copy of their instrument. This instrument, as you see, has a distal lateral opening. The trocar, upon a wire, comes through the proximal side arm. Readings are obtained through a needle, only after one notes the presence of pleural symphysis. The needle is thrust into the first intercostal space. This space, although not the only point of entrance used, is, in our experience, the most usual location for instrumentation.

(Slide) The depth to which the "sounding" needle must be thrust in order to enter the pulmonary cavity is a very exact question. Anteroposterior and lateral films are of value as are tomographic exposures. However, they may be confusing. As I have just indicated, one notes that, in the majority of cases, the center of the cavity is in a horizontal plane with the first anterior intercostal space. Minor

*See page 485 for article by Brunn, Shipman, Goldman, and Ackerman.

studies at this level were made. We found that the average distance between the anterior chest wall and the posterior chest wall is about 12.5 cm. The average distance between the anterior chest wall and the anterior wall of the cavity is about 7 cm.

(Slide) Because of our desire to inspect the cavity and because of our desire to cauterize the bronchi, we were forced to modify definitely the Monaldi instrument. Here is the cannula. Here is the trocar which has the opening or vents at the distal spear-like part. Here is a flange which is used to keep the cannula in place. This is a permanent affair. No rubber catheter is initially used.

(Slide) This slide shows the instrument through the chest wall. This is the rubber disc lying between the flange and the chest wall and here is the flange which, with adhesive over it, stabilizes the instrument and keeps it firmly in place.

(Slide) These are the telescopes which are used.

(Slide) This is the material which was aspirated from the tuberculous cavity. Note the amount which was obtained over a period of two and one-half days.

(Slide) Here is a special glass valve and bottle which I use.

(Slide) The height of the water or the mercury in the inner valve determines or regulates the amount of negative pressure you are using, regardless of the amount of pressure furnished by drawing water or electric suction device.

(Slide) Here is a patient with a definite pulmonary cavity.

(Slide) Here is the instrument in place.

(Slide) Here you note a so-called ascending bronchogram.

(Slide) I merely show you this specimen for one particular reason, because this procedure of intracavernous aspiration is not without danger. Note the Rasmussen aneurysm. Because of this, one is cautioned to avoid the medial aspect of the pulmonary cavity for it is here that one finds the Rasmussen aneurysm if it is present.

(Slide) This is the artist's representation of what one sees in the pulmonary cavity. In a moment I shall show you an actual colored photograph of the walls of a cavity in the living, taken through the telescope.

(Slide) Here is a recess which the artist noted. It is commonly found posteriorly. In the early stages, the walls are granular in appearance and roughened, and the surface of the walls is a grayish-brown. The detritus is thrown off, the walls become smooth and glistening; and finally one notes capillaries. Now there is a definite redness to be seen. The cavity wall has a very healthy, fleshy appearance.

These are the colored photographs. Here is a recess in the cavity wall which you will note is posterior.

(Slide) Here is the bronchus which leads to the cavity.

My only conclusive thought in this particularly stimulating question is that I do not know what the Monaldi technique will accomplish. I am not overenthusiastic about it as a procedure per se. However, I am markedly enthusiastic about the possibilities it furnishes in the study of the biology and mechanics of pulmonary cavities.

DR. ARTHUR VORWALD, Saranac Lake, N. Y.—I was glad to hear Dr. Shipman speak of total occlusion of the bronchus. It seems to me that, in considering the draining bronchus and its relationship to the cavity in tuberculosis, the words "total" or "partial" should complement the term bronchial stenosis, for the clinical signs and symptoms and the course of the cavity depend greatly upon whether the disease in the bronchus brings about a partial or a complete occlusion of that bronchus.

As regards the suction applied in the Monaldi procedure, there is one question that occurs to me. In view of the rich capillary system in the granulation tissue zone that makes up the wall of the cavity, is it not possible for suction to produce uncontrollable hemorrhage?

In closing, I wish to leave this thought with you. The course of a cavity in tuberculosis is not entirely dependent upon the changes in the bronchus. The inflammatory reaction in the wall of that cavity determines in large part what will happen to the lesion.

DR. J. D. STEELE, Milwaukee.—I should like to present one case of open cavity drainage which was performed in preparation for thoracoplasty.

This girl had a huge tension cavity (15 by 8 cm.) which had been present for two years. It was proved to be a pulmonary cavity. It was drained, and six months later, without suction, it had decreased to this size (10 by 5 cm.).

Subsequently the cavity did not decrease further in the course of four months. An eight-rib posterior thoracoplasty was performed, and lipiodol was injected into the cavity six months after thoracoplasty. This was taken six weeks ago (cavity now 3 by 1 cm.). There was very little oil present, and it doesn't show on the film. However, within the last two or three weeks the sinus has apparently closed.

DR. ALFRED GOLDMAN, San Francisco.—I should like to give the report of one case and show the kymographic tracings of intracavitary pressures which we made on Dr. Harold Brunn's service at the University of California.

(Slide) This is the photograph of a patient who had a bilateral transpleural decompression, or modified Monaldi method of intracavitary drainage. It shows the apparatus used to apply suction. In this case, however, suction was not applied for several months, but the cavities were exposed to atmospheric pressure only. This catheter goes into the cavity on the left side, and this into the one on the right. The tubes join here, and this little motor furnishes the constant suction.

(Slide) This is the patient's roentgenographic history of the cavity on the right side. In September there was a large thin-walled cavity in the right upper lobe, and a metal trocar with about a quarter-inch bore was inserted on October 16. The cavity was not sucked upon, but it diminished in size, and the same thing that Dr. Ornstein mentioned, the apparent atelectasis, occurred around the cavity as it shrank.

This is the ascending bronchogram made two and one-half months after the transpleural decompression, at a time when the secretions from both cavities, as well as the sputum, had become negative for tubercle bacilli. A small narrowed bronchus is outlined, leading from the cavity.

This roentgenogram was taken about six weeks later, showing the same cavity, still very small and still negative for tubercle bacilli, and the bronchus still leading from the cavity. The secretions from these cavities became negative for tubercle bacilli while the cavities could still be seen. Kupka and others have noted the same finding. Whether or not the bronchi are occluded or whether or not suction is applied, the tubercle bacilli disappear from the secretions.

(Slide) This is a kymographic tracing of intracavitary pressures made on the day that the left cavity was drained with a cannula. These pressures are made simply by connecting the cannula with a writing tambour and a water manometer. The tracings show that, when we artificially decrease the pressures in such a cavity, these pressures are maintained at the decreased level. Thus, at this point, 50 c.c. of air were sucked out of the system and pressures were reduced to –1, –10 and were maintained at this level, showing that there is no real aeration of the cavity through its bronchus. When the pressures were reduced further by removal of an additional 25 c.c. of air, a negative pressure was reached at which we observed the entrance of air into the cavity; this was announced by a wheeze and an elevation of the pressures so that they became less negative, rising from –12, –4 to –2, –6. We then raised the pressures artificially to +12, +4, and they were stabilized around +8, +2 where they were maintained.

These studies, we think, demonstrate valvular effects. When we speak of a valve, we mean some mechanism which controls the air flow in one direction. In this instance, we feel that the air flow is more prominent in the direction from the bronchus to the cavity and that the air tends to be entrapped in the cavity. In Dr. Eloesser's pictures, it is evident that the intracavitary pressures never became less negative during the artificial breathing of the cadavers, but always became more positive. This indicates a bronchus-to-cavity valvular relationship which works to entrap the air in the cavity and not to allow the air to flow in one direction out of the cavity.

DR. R. M. LEWIS, Philadelphia.—As Dr. Davidson said, I think we should all feel quite overwhelmed with all the information that has come over the board this afternoon. I think the most important thing that has been said today, or that has been done, is that all the work presented points to a common understanding of a definite mechanism.

I shall start with the last part of my discussion, that is, the last slide that Dr. Goldman showed. In this case he said there were no subsequent pressure changes when he insufflated or aspirated the cavity. Could we have that slide again? (Dr. Goldman's slide.) If you notice when the aspiration is begun, the pressure is low but gradually it rises toward the base line, and, after insufflation is stopped with the pressure above the base line, there is a gradual fall toward the base line. Therefore, air passes through this bronchus. You notice, that after he aspirates, he gets a negative pressure in his cavity. As the respiration goes on, the general slope of that respiratory curve is up toward the atmospheric base line. When he insufflates the cavity and raises the pressure, the general slope of that curve is downward toward the atmospheric base line. That means to me that there is a flow of air through the open bronchus, but obviously at a low velocity.

In Dr. Eloesser's descriptions, we notice that sometimes there was more rapid flow of air through the bronchus than there was at other times, but we must also remember that, if air was flowing, something was causing it, and that something was an entirely different pressure interchange.

When one talks about a ball valve in the bronchus, I think it leads us astray. There is no ball valve so far as anybody has demonstrated. I have never seen evidence of a ball valve in the true sense of the word. There is definitely an open bronchus which at times is obstructed. The change in shape of the lung or some substance, such as the exudate in the cavity, getting into the bronchus or by fibrous overgrowth which involves the bronchus may partially block the easy flow of air from the cavity. These are changes which may gradually occur in the case of fibrosis and may occur intermittently in the case of exudates in the bronchus.

There has been a definite correlation between Dr. Goldman and Dr. Eloesser. Dr. Goldman introduced his air from the outside into the lung, and then into the cavity. Dr. Eloesser introduced air through the trachea, through the bronchus into the cavity. Both tie up definitely with relative pressures.

To go back to the ball-valve mechanism, we must consider the partial obstruction of the bronchus more in the light of the valves in the cystic duct. It is on a tortuous channel which changes shape with change in respiration. We have a simple correlated mechanism of pressures based on absolute pressure rather than atmospheric pressure. Gases flow from a higher to a lower pressure in an attempt to equalize.

In the pressure changes that were noted when the discussion of Dr. Steele was on, he intubated his large cavity. In intubating his cavity, he merely said, "We will now introduce air at atmospheric pressure into the cavity, and, if more air comes in through the trachea and subsequently the bronchus into this cavity, it will flow out through the tube we have put in. Therefore, we have made the pressure

in there atmospheric; it can never become more and it can never become less, and, if there is actual flow out of the cavity, it is the result of the intrinsic collapse of the lung tissue itself.''

So, if we look at our pressures as differential pressures and evaluate them in their true light, I think we shall gradually get to a more thorough understanding of the mechanics involved in cavity closure.

DR. SIDNEY SHIPMAN, San Francisco.—I suggested that Dr. Brunn might close this discussion because really he has been the mainspring at the hospital in San Francisco where we have been led to do some of these things, but he does not seem to be present now.

I have nothing to say in closing except that I appreciate very much what has been said here and the light that has been thrown upon this question of cavitation. Otherwise we certainly are very much confused.

I should like to thank Dr. Alexander for making a remark a moment ago to me. He said he hoped nobody would go away with the idea that thoracoplasty was out of date. We certainly did not intend to convey anything like that, but only to point out that in certain cases where thoracoplasty fails perhaps we do need a little different approach.

DR. LEO ELOESSER, San Francisco.—I am not sure whether this discussion has clarified or confused the ideas of the audience. What I tried to show was that no absolutely permanent mechanical condition obtains in many cavities, such as obtains in a water pump, but that they are under positive pressure for a considerable amount of time, that for *most* of the time it is easier for air to be entrapped in them than it is for air to get out, and that, therefore, one should consider intracavitary pressure factors also, rather than pulmonary pressure factors alone.

Also, to summarize, I should like to advocate needling cavities wherever it is possible; that is to say, when one is quite sure that no pneumothorax is possible or exists, I should advise that cavities be needled in an attempt to study the conditions that pertain in them before thoracoplasty is done. The needling is harmless. We have done it frequently without any upset. I think some patients may be saved from thoracoplasty by this procedure, or possibly a thoracoplasty may be done in a different way from which we would originally intend, without knowledge of intracavity pressure.

I agree with Dr. Alexander that the day of thoracoplasty is not over. However, I have a lurking suspicion that we have been more or less on the wrong track and that our attempts to obliterate pulmonary cavities anatomically by increasingly wide rib resections, by extensive resections of the transverse processes, of the scapula and what not, may often lead to failure, that a purely anatomic viewpoint has led us to neglect the physiologic or pathologic one, and that the results observed by Brunner in his early summary of Sauerbruch's cases still obtain; that is, if you are going to get a good result, you get it (or begin to get it) at your first operation. If you don't get it then, you don't get it at all.

DR. W. A. HUDSON, Detroit.—I had hoped that Dr. Lambert would proceed with the next papers without calling upon me for this discussion. But I am quite happy to discuss Dr. Kupka's paper, first, because I have had the pleasure of having Dr. Kupka work with me; second, because, while he was in Rome, I was in touch with him and he very kindly kept me posted on what he was seeing there, and after his return I spent a very pleasant evening going over the work he had done while in Rome. On his first presentation of this work to me, I was very favorably impressed. The interpretation that he put upon what he had seen seemed very reasonable. This procedure seemed to afford us the means of treating a certain group of people for whom we had been unable to do something previously.

In other words, in the past we might say from 60 to 85 per cent of the patients entering the sanatorium were permitted to have some form of collapse therapy. There remained, however, that other group who were not considered suitable for thoracoplasty, pneumothorax, pneumonolysis, or phrenic, and they were left to lie in bed and wait for whatever came their way. Now it seemed to me we had another attack we could make upon the disease. Tuberculosis workers are a hopeful lot of individuals, and I think the thoracic surgeon is probably the most optimistic of them all. If he were not optimistic, he would not be continually hammering away trying to do something for this particular group of people.

The introduction of intracavity suction drainage offered an opportunity to remove secretions, as Dr. Kupka told you. It offered an opportunity to prepare the patient, if possible, for further surgery, whatever that might be. We have chosen in our cases to apply intracavity suction drainage to a group of patients who were entirely, to our way of thinking, out of bounds as far as the ordinary collapse measures were concerned. They had for the most part been in the sanatorium for long periods. I will show you three cases to illustrate the type that we have used.

(Slide) The first patient is a man who had refused thoracoplasty. He had a chronic case with a cavity at the apex, positive sputum, and disease in the opposite lung. We might have considered him for thoracoplasty, but the patient would not consent.

(Slide) The catheter was inserted on the fifth day of January this year. This is a film made immediately after insertion of the catheter. You will note that the catheter has been inserted in such a manner that it completely outlines the cavity. This catheter was inserted from the front through the first interspace. Manometer readings were made at the time, and secretions were taken at the time the catheter was inserted and were found to be positive. In a short period of time the patient's sputum was negative and his secretions were negative.

(Slide) This film, made on the 29th of the past month, reveals the catheter track, and there has been aeration of the adjacent lung tissue. At first, it was suggested that the increased density about the cavity represented an increasing atelectasis. I do not believe that this is true. It is my impression that with the establishment of suction there occurs at first a definite increase in the vascularity of the tissue immediately adjacent to the cavity, that, as the drainage becomes effective, there is a definite movement of tissue fluids from the zone of inflammation toward the cavity, and that in time this movement of fluids through the tissue is the means by which the products of inflammation are carried into the cavity and thence out through the drainage tube. When one is dealing with a cavity the size of the one we started out with in this case, and with the extensive and heavy scarring that is present in the cavity wall, it seems unreasonable to expect to produce sufficient suction against that heavily scarred wall to draw the walls together and hold them together until union between those cavity walls has taken place. We have therefore introduced the use of certain solutions to wash out the secretion, and we have used digestive solutions (enzymol) to digest the rotten tissue that lined the walls and projected into the crypts. We are convinced from our observations that these procedures are distinct assets in the closure of cavities such as the one just presented.

(Slide) We now present a patient with a very large cavity which contains a fluid level. The thickness of the wall of this cavity is measured in centimeters—not in millimeters. It is heavy and stiff like leather. There is no reason why we should expect these walls to come together and unite permanently any more than we should expect a chronic fibrotic parietal pleura to unite with fibrotic visceral pleura. In those cases we have in the past done Schede operations. In this case we have inserted a catheter as shown in this slide.

(Slide) The catheter is in place, and you will note that this represents the contour of the cavity, which is roughly of a dumbbell shape, with the tip of the catheter projecting into the upper extension of the cavity. There has been some immediate change in the contour of the cavity on the introduction of the catheter. Inspection of this cavity at the time of the operation, by means of a thoracoscope inserted through the trocar, revealed some very interesting facts. The wall of the cavity was made up of a heavy scar, thoroughly impregnated with decayed tissue; there was fluid in the bottom of the cavity and numerous projections of fibrotic bands into the cavity and across the cavity.

(Slide) In presenting this additional case, we call your attention to the enormous size of the cavity. Inspection of the cavity through the thoracoscope showed bands projecting across the cavity and ridges of scarred and necrotic tissue projecting into the cavity. Numerous pockets and crevices were noted, all filled with necrotic tissue and pus. It did not seem reasonable to expect this cavity to close with all that dead material in it. The use of our digestive solutions has helped to remove that dead tissue (slide) as shown in this slide. It is noted that in a matter of a few weeks there has occurred a distinct change in the contours of the cavity. During this time the cavity has not closed, but with removal of this necrotic tissue there has been a distinct improvement in the patient's general condition. At the time the catheter was inserted, it was necessary to lift the patient from the stretcher to the x-ray table. The nurse washed her hands and fed her. She now is able to remove herself from the stretcher to the x-ray table. She feeds herself, and she is bacilli free as far as her secretion is concerned.

Incidentally, this brings up another point. Somebody said "What happens to these people if you put too much pull on blood vessels within the cavity wall?" The third day after I inserted the catheter in this woman, she had a massive hemorrhage. They called me and asked what to do with it. I said, "Leave the catheter in place, and treat her just the same as though she had had an ordinary hemorrhage." Then after a couple of days, we went ahead with our suction. I feel we were justified in this case.

We feel that this procedure definitely offers us a new attack on a group of tubercular patients in whom we have found our hands tied in the past. I do not believe that anybody can give us the answer as to what the ultimate application of this proceeding will be, but I believe it is a perfectly logical and a very promising procedure and that it should be carried further and the answer determined.

Particular credit should be given to Dr. Earl Bullock for his timely suggestions, helpful criticisms, and constant attention in the handling of this group of patients.

CORONARY SCLEROSIS AND ANGINA PECTORIS

Report of Thirty Patients Treated by the Beck Operation

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THE purpose of this paper is to present a follow-up report on the patients who had vascularized grafts placed upon the heart for the purpose of producing a collateral blood supply to the heart. A clinical report has not been made since 1937. The first of these operations was done Feb. 13, 1935; the last, April 19, 1938. Thirty patients were operated upon. Up to the present time, the data are incomplete. We have considerable clinical information concerning results, but we lack a sufficient number of specimens in which the blood vessels were injected because the patients continue to live.

All patients were carefully studied and the diagnosis of coronary sclerosis was definitely established. Further, all patients were given the usual medical therapy for angina pectoris, but despite therapy, their tolerance for physical and emotional strain was greatly reduced. Fourteen of the patients had had one or more attacks of coronary thrombosis previous to the operation (electrocardiographic or pathologic evidence).

In this group of thirty patients (twenty-nine males, one female) there were no immediate operative deaths. The total mortality was 33.3 per cent (ten patients), but the mortality of the first fifteen cases was 47 per cent (seven patients), while the mortality of the last fifteen cases was 20 per cent (three patients). Table I is a summary of the patients not surviving the operation and the causes of their deaths.

There were three deaths caused by arterial thrombosis. In view of this complication, every effort was made to avoid dehydration, anoxemia, and fall in blood pressure. In one patient (Case 7), auricular fibrillation with rapid rate was followed by a fall in blood pressure. Cerebral thrombosis (or embolism) resulted in right hemiplegia and death. In Patient 8, a nonoccluding mural thrombus was found in the oblique branch of the left coronary artery. There were three cases of sudden death, all in patients with severe generalized coronary arterio-

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sclerosis. No recent thrombosis was found. Three patients had bronchopneumonia, and mediastinitis was the cause of one death.

TABLE I

PATIENT	SURVIVAL PERIOD (DAYS)	CAUSE OF DEATH
2	6	Thrombosis of abdominal aorta
5	2	*B. Welchii* mediastinal infection
7	4	Cerebral thrombosis, bronchopneumonia
8	1	Coronary thrombosis
10	2	Sudden death; aneurysm posterior left ventricle
12	9	Pneumonia
14	5	Pneumonia
16	1	Sudden death; advanced coronary artery disease
29	2	Pneumonia and pleural effusion
30	1	Sudden death; advanced coronary artery disease

Survival Cases.—Twenty patients survived the operation and were able to leave the hospital. Table II is a summary of these cases.

TABLE II

CASE	DATE OF OPERATION	PERIOD OF SURVIVAL			CAUSE OF DEATH	CLINICAL RESULT OF OPERATION
		YR.	MO.	DAYS*		
1	2/13/35	5	3	18		Good
3	6/28/35	2	5	29	†Inanition; Simmonds' disease	Poor
4	7/ 9/35	4	10	22		Fair
6	8/31/35	4	9	0		Good
9	1/ 6/36	4	4	25		Good
11	2/22/36	4	3	9		Good
13	5/25/36		5	29	Cardiac failure; myocardial degeneration	Poor
15	7/25/36	3	10	6		Good
17	11/18/36	3	6	13		Good
18	11/21/36	3	6	10		Good
19	12/ 3/36	3	5	28		Good
20	12/ 5/36	2	2	26	†Cerebral hemorrhage	Good
21	1/30/37	3	4	1		Good
22	1/30/37	3	4	1		Good
23	3/ 6/37	3	2	25		Good
24	4/ 9/37	1	2	25	†Coronary thrombosis	Poor
25	4/24/37	3	1	7		Fair
26	7/30/37	2	10	1		Fair
27	8/ 4/37	2	9	27		Fair
28	8/13/37	2	9	18		Good

*Time as of June 1, 1940.
†Post-mortem examination performed.

Survival Cases Dying Remotely Following the Operation.—The third patient in the series died two and one-half years after operation of inanition, with a clinical picture resembling Simmonds' disease. He had had a total thyroidectomy one year prior to the operation on the heart. He was able to take $\frac{1}{10}$ grain of thyroid extract daily for the relief of the symptoms of hypothyroidism, and he had little pain. Previous to the operation this small amount of thyroid extract had increased the pain.

The twentieth patient was strikingly improved by the operation and was able to return to his occupation as a salesman. He died two years and three months later of cerebral hemorrhage.

The twenty-fourth patient died one year and three months following the operation of myocardial infarction. His postoperative course was not marked by great decrease in the angina. The pathologic changes in the coronary arteries were severe and extensive and the lack of improvement is readily explained.

Special Cases.—One patient who had early cardiac failure (Case 13) due to hypertensive and coronary artery disease was operated upon. Because of his circulatory failure, the complete operation was not performed, bone powder only being placed within the pericardial cavity. The patient survived for six months and died of congestive failure. Post-mortem examination was not permitted. Two patients with hypothyroidism were operated upon, one following total thyroidectomy and the other a case of spontaneous myxedema. The first patient (Case 3) is described in the preceding paragraph. The second, a patient with spontaneous myxedema (Case 17), was unable to take thyroid extract preoperatively, but after the operation was relieved of his symptoms of myxedema by the taking of thyroid extract. Another patient (Case 12) had a supposed total thyroidectomy performed seven months previously, but he had no clinical evidence of hypothyroidism. He was not relieved by the thyroidectomy. Another patient (Case 21) had had alcoholic injection of the cervical sympathetic ganglia, without relief. Following the cardiac operation, striking relief of symptoms occurred, and he has been able to return to his work. Many of the patients had generalized arteriosclerosis and the postoperative complications in Cases 2 and 8 were directly due to this pathologic change.

Study of Possible Cardiac Compression as the Result of the Postoperative Scar.—In ten patients studies were carried out to determine whether chronic cardiac compression followed the operation. Venous pressure and kymograph roentgenograms were taken. In only one patient was the venous pressure elevated to 15 cm. of physiologic solution of sodium chloride. In the other patients the venous pressure at the elbow measured from 3 to 11 cm. Study of the roentgenograms revealed no diminution in the pulsation of the heart as recorded by the depths of the notching of the cardiac silhouette. Signs of chronic compression of the heart were not found in any of the patients after this operation was performed.

Electrocardiographic Observations.—In sixteen of the patients (53.3 per cent), the electrocardiogram at rest was abnormal, the chief changes being inversion of T_1 or T_2, or both, notching of R_1 or R_2, or deep Q_2 and Q_3. In twenty-three instances (76.6 per cent), there were S-T deviations after the exercise test (see Table III). In one additional patient, negativity was observed during the angina, bringing up the

total of abnormalities to twenty-four, or 80 per cent of all the patients operated upon. Return of the electrocardiogram to normal was not anticipated after the operation, as the underlying myocardial fibrosis is not a reversible process. In no case was there return of the electrocardiogram to normal, and many of the patients had postoperative changes due to the operative trauma of the superficial myocardium.

TABLE III

ELECTROCARDIOGRAMS

CASE	AT REST								S-T DEVIATION AFTER EXERCISE							
	BEFORE OPERATION				AFTER OPERATION				BEFORE OPERATION				AFTER OPERATION			
Lead	I	II	III	IV	I	II	III	IV	I	II	III	IV	I	II	III	IV
1	N	N	N	N	T–	T–		T–					TO	TO	TO	T–
2	N	N	N	N	D				S-T–	S-T–	N	S-T–	D			
3	N	N	N	N	N	N	N	N								
					T–	T–		T–								
4	T±	Q	Q	N		Q	Q			Q	Q		T+	T+		S-T–
5	N	N	N	N	D				S-T–			S-T–	D			
												+				
6	N	N	N	N	N	T–	N	N	·	S-T–		T–				
7	N	N	N	N	D				S-T–	S-T–		S-T–	D			
									S-T–	S-T–		S-T–				
8	N	N	N	N	D							T–	D			
9	N	N	N	N	T±	T–	S-T+	T–				S-T–	S-T–	S-T–	S-T+	T–
		Q	Q													
10	N	T–	T–	N	D				S-T–	S-T+	S-T+	S-T–	D			
11	N	N	N	–	N	N	N	N								
				Q						S-T–	S-T–	T–				
12	RN	RN	RN	T–	D				T–	T–	T–	S-T–	D			
13	T–	Q	Q	T–					S-T–				T			
	Q			Q												
14	T–	N	N	T–	D					S-T–	S-T–		D			
												S-T–				S-T–
15	N	T–	N	N	T–	T–	N	T–				T–	S-T–	S-T+	S-T+	T–
16	N	N	N	T–	D				S-T–	S-T–		S-T–	D			
17	TO	TO	TO	TO	T–	N	N	T–	S-T–	S-T–	S-T–	S-T–	S-T–	S-T–		S-T±
													S-T–			S-T+
18	T–	N	N	T–	T–	N	N	T–		S-T–			T–	S-T–		T+
		RN	RN		RN	RN	RN									
19	TO	Q	Q	N		Q	Q	Q								
						T–	T–					S-T–				
		T–			T–			T–								
20	ALA				ALA				S-T–	N	N	S-T–				S-T+
21	N	N	N	N					S-T–	S-T–	S-T–	S-T–				
22	N	N	N	N					TS	TS	TS	TS				
23	N	N	N	N	N	N	N	T–		S-T–	S-T–	S-T–		S-T–	S-T–	T–
		Q	Q	Q		Q	Q	Q		Q	Q	Q				
24	N	T–	T–	T–	N	T–	T–	T–	–	T–	T–	T–	T+ +			+ +
25	N	N	N	N	T–	N	N	T–	T–			T–	S-T–			T–
26	N	T–	T–	T–	T+	N	N	T–	S-T–	S-T–		S-T–		T–	T–	T–
27	N	N	N	N	N	N	N	S-T–				–				
								Q								
28	T–	T–	Q	T–	T–	T–	Q	T–	–			+	–	–		+
29	N	N	N	T–	D							S-T–	D			
		Q	Q													
30	N	T–	T–	S-T–	D				S-T–	S-T–		S-T–	D			

N, Normal
D, Died
TO, T isoelectric
T+, T slightly positive
T–, T slightly negative
TS, T smaller
RN, R notched
ALA, Abnormal left axis deviation
Q,
Blank, No change

REPORT OF PATIENTS WHO SURVIVED

CASE 1.—Arteriosclerotic heart disease and angina pectoris of nine years' duration, severe for five years; moderate generalized arteriosclerosis with hypertension; arterial pressure systolic 164, diastolic 92 mm. Hg; moderate chronic pulmonary emphysema. A 48-year-old farmer, with gradually diminishing exercise tolerance, finally became incapacitated for any work because of pain. The exercise tolerance test, sixty trips on the steps, brought on precordial pain, fatigue, and dyspnea. Operation was done Feb. 13, 1935. Four months later he began to do light work around the hospital as a gardener and made eighty-two trips on the steps without pain but stopped because of dyspnea. The patient has been a farmer for four years, working twelve hours daily. He feels no more than the usual fatigue at the end of the day. The result in this, the first patient to have the operation, was excellent.

CASE 3.—Arteriosclerotic heart disease and angina pectoris for five years; severe myxedema from total thyroidectomy in 1934. A business man, aged 51 years, with severe angina of effort and of emotion, became totally incapacitated. The exercise tolerance test, fifteen trips on the stairs, produced complete exhaustion. Operation was done June 28, 1935. The amount of thyroid extract that the patient was able to take without producing pain after operation was two or three times the amount taken before operation. Tolerance for exercise improved only slightly. The patient believed that the operation lessened the pain a great deal, but he remained weak. The subsequent course was unsatisfactory. Although his symptoms of hypothyroidism were controlled by thyroid extract (0.1 grain daily), he became emaciated and extremely weak. The clinical picture resembled that of Simmonds' disease. Anterior pituitary extract was given hypodermically. He died Dec. 27, 1937. A post-mortem examination was performed. The best that can be said for the postoperative results in this patient is that he was largely relieved of pain despite adequate administration of thyroid extract for the symptoms of myxedema following total thyroidectomy.

CASE 4.—Arteriosclerotic heart disease, coronary sclerosis, and angina pectoris of five years' duration; generalized arteriosclerosis and diabetes mellitus. A surgeon, aged 50 years, with angina of effort and emotion, was totally incapacitated in December, 1932. Subtotal thyroidectomy was done in January, 1933. He had a coronary thrombosis in February, 1933. The patient had had diabetes mellitus since 1918, requiring insulin. He had had pain in the legs for three years on walking. A roentgenogram of the legs showed calcification of the vessels. The electrocardiogram showed a deep Q_3. Operation was done July 9, 1935. The exercise tolerance has increased from eighteen trips before operation to thirty-two trips. The patient has less pain on exertion and on exposure to emotional strain. Four years and ten months have now elapsed since the operation, and pain rarely occurs. He is able to walk as much as four blocks without pain and is prevented from taking more exercise by his peripheral vascular disease. The patient believes that his symptoms have been greatly relieved and that his tolerance for physical exertion and emotional strain has been definitely improved. The results in this case are classified as fair.

CASE 6.—Arteriosclerotic heart disease and angina pectoris for two and one-half years; generalized arteriosclerosis and intermittent claudication. A machinist, aged 55 years, had the typical anginal syndrome brought on by exercise or emotion. The symptoms began in April, 1933, after a severe attack of pain due to coronary thrombosis. He returned to work, but tolerance for exercise progres-

sively decreased and in March, 1935, he became totally incapacitated. The electrocardiograms showed a reversal of the T wave in the chest lead, and after exercise there were the typical changes seen in coronary arteriosclerosis. Operation was done August 31. The patient returned to his former job five months after operation. Exercise tolerance increased from twenty-four trips before operation to thirty-seven trips after operation, and the patient had to stop the step test, not because of anginal pain, as before the operation, but because of claudication. Because of these symptoms in his legs, the patient changed his job in July, 1936, to that of a storekeeper. The patient has no anginal pain and is now working as a watchman. In this case of generalized arteriosclerosis with a remote myocardial infarct, the result is excellent.

CASE 9.—Arteriosclerotic heart disease and angina pectoris for three years and three months. A painter, aged 45 years, was forced to reduce his activities and finally was totally unable to work for two years and three months. Angina was produced by seventy trips on the steps, and the electrocardiogram, which at rest was normal, showed characteristic changes in the S-T interval. Glyceryl trinitrate increased the exercise tolerance to 102 trips. Operation was done Jan. 2, 1936. A year later he returned to his former work as a painter and was able to do a good day's work, with little discomfort. His ability to walk has not increased in proportion to his ability to take other exercise. He can make fifty-two trips on the steps without pain and without glyceryl trinitrate. It is now four years and four months since the operation, and the patient has been restored to economic independence. On walking long distances, he still has a recurrence of pain, but he has sufficient reserve to enable him to work a full day.

CASE 11.—Arteriosclerotic heart disease and angina pectoris for sixteen months; probable coronary thrombosis at onset of illness. A tailor, aged 42 years, had angina of effort and emotion. He became totally incapacitated. Rest and drugs failed to give relief. Angina was produced by twenty-four trips on the steps. After glyceryl trinitrate he was able to make forty-six trips before anginal pain was felt. The conventional electrocardiogram was normal, but T wave in the chest lead was reversed in direction. Operation was done Feb. 22, 1936. The patient is now able to work. He can tolerate ninety-seven trips on the steps and stops because of dyspnea and not because of pain (Fig. 1). He is comfortable most of the time and rarely takes nitroglycerin.

CASE 13.—Arteriosclerotic heart disease and angina pectoris for eleven years; probable attack of coronary thrombosis six years before admission; generalized arteriosclerosis and hypertension; early cardiac failure. A man, aged 58 years, an accountant, had dyspnea and edema of the ankles, which had appeared recently. The heart was enlarged. The electrocardiogram showed regular sinus rhythm, splintering of QRS in all leads, and inversion of T in Leads I and II; T wave in the chest lead was reversed. The circulation time was 20 seconds. Because the patient was showing signs of heart failure, the usual operation was not done. On May 25, 1936, one costal cartilage was removed; the pericardial cavity was opened and powdered beef bone was placed within. The congestive failure responded to treatment, but four months later the patient died. There was no autopsy.

CASE 15.—Arteriosclerotic heart disease and angina pectoris for five years. A salesman, aged 48 years, could not use nitrites because these drugs produced headache. He had restricted his activities to a minimum in order to prevent pain. The electrocardiogram showed inversion of T_2 and displacement of S-T in the chest

lead beyond the normal limit. The left ventricle was enlarged. Operation was done July 25, 1936. Some improvement followed, and the patient returned to work. Exercise tolerance has continued to increase. He is exceedingly active, walking up and down stairs without symptoms. Unusual emotional strain still brings on pain occasionally.

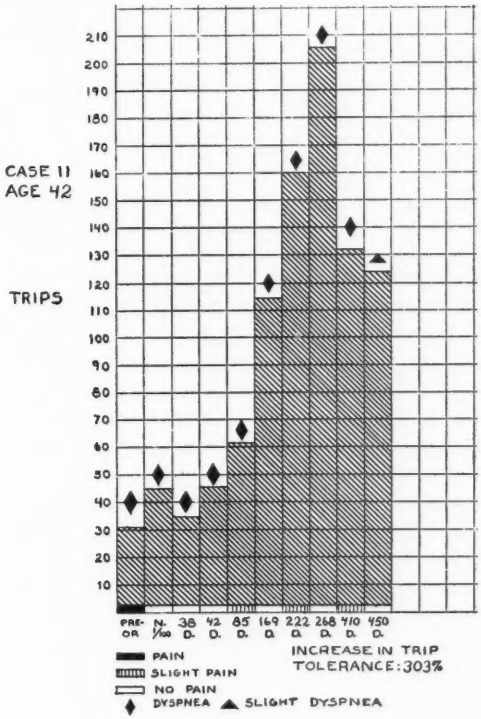

Fig. 1.

CASE 17.—Arteriosclerotic heart disease and angina pectoris; myxedema and secondary anemia. A machinist, aged 50 years, had had a typical history of angina of effort for eighteen months. The basal metabolic rate was −30, and the patient took small doses of thyroid. The electrocardiogram revealed a left axis deviation and QRS of normal voltage; the T wave was almost isoelectric in all leads, including the chest lead. Operation was done Nov. 18, 1936. Congestive heart failure developed after operation and cleared up with appropriate treatment. Tolerance for exercise increased from thirty-six trips on the stairs before operation to fifty trips after operation. The dose of thyroid extract has been increased to ¼ grain. He does not take any nitroglycerin. He has been working at his job as machinist much of the time, laying off only when there is no work available.

CASE 18.—Arteriosclerotic heart disease with angina pectoris of nine months' duration. A Greek waiter, aged 48 years, had been incapacitated for work for six months. The T wave in Lead I of the electrocardiogram was inverted; in the

chest lead it was also reversed. The left ventricle was questionably enlarged. Operation was done Nov. 21, 1936. Congestive heart failure developed after the operation. The patient has shown gradual but slow improvement. He is now working. He takes an occasional tablet of glyceryl trinitrate. Before operation, he used five or six tablets daily. It is now three years and six months since the operation, and he is able to carry on a full day's work without symptoms, either as cook or as cashier in a restaurant. On walking five or six blocks, moderate dyspnea develops. At the present time he has no evidence of cardiac failure, and the circulation time has decreased from 27 seconds to 15.6 seconds.

CASE 19.—Arteriosclerotic heart disease and angina pectoris of seventeen months' duration. A coal miner, aged 49 years, was totally incapacitated during the entire course of his illness. He had severe pain at rest, and morphine was given for the pain. Theophylline with ethylenediamine and glyceryl trinitrate gave no relief. The electrocardiogram showed alterations indicative of a remote posterior and basal infarct. Operation was done Dec. 3, 1936. Marked improvement followed operation, but pain now occurs occasionally.

CASE 20.—Arteriosclerotic heart disease and angina pectoris for one year. A salesman, aged 69 years, presented the typical symptoms of angina pectoris and became totally incapacitated. There was no episode suggestive of coronary thrombosis. The left ventricle was slightly enlarged.. The arterial pressure was 150/100. Calcification of the peripheral arteries was present. The electrocardiogram showed regular sinus rhythm, left axis deviation, Q_1 and Q_2, and a normal chest lead.

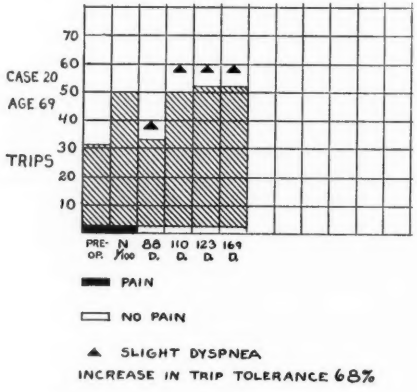

Fig. 2.

After exercise the chest lead showed the S-T portion sharply elevated (upright deflection positive in value). The circulation time was 25 seconds. Operation was done Dec. 5, 1936. The patient became free from pain and discontinued all treatment. His activities are increased (Fig. 2). The result is excellent. He could walk with comfort and returned to his work, walking much of the day; all medication was stopped and there was no recurrence of pain. His exercise tolerance as measured by the two-step test has remained about fifty trips. He continued at his work until March 1, 1939, when he suddenly developed a cerebral hemorrhage, from which he died, two years and two months following operation.

CASE 21.—Arteriosclerotic heart disease and angina pectoris of five years' duration; mild diabetes mellitus. A clerk, aged 51 years, failed to benefit from med-

ical treatment; paravertebral injection with alcohol failed to give relief and he became totally incapacitated for work. The left ventricle was slightly enlarged. The peripheral arteries were calcified. The electrocardiogram was normal at rest but after exercise showed definite alterations from normal. Operation was done Jan. 20, 1937. The patient was much improved following operation. The pain has not completely disappeared, but it is less severe. He has returned to work.

CASE 22.—Arteriosclerotic heart disease and angina pectoris; thromboangiitis obliterans. A cook, aged 42 years, had angina of effort and emotion for five and one-half years and was incapacitated for his work for two years. He took morphine for the pain. The left foot was cool, and the arterial pulse was not palpable in the left leg. The electrocardiogram showed no significant changes before or after exercise. Operation was done Jan. 30, 1936. He had several anginal attacks in the early postoperative period. Later he showed striking improvement. He is up and about and does not require medication. The interesting question arises whether the disease of the coronary arteries bears any relationship to the thromboangiitis obliterans of the leg.

CASE 23.—Arteriosclerotic heart disease and angina pectoris of seven months' duration. A molder, aged 49 years, had a typical history of angina of effort for seven months and had had to give up work four months before admission. Medical care with a long rest in bed did not increase his tolerance for exercise. The anginal syndrome was produced by fifty-three trips, and after glyceryl trinitrate he was able to make ninety trips. Electrocardiographic changes were present during the induced angina. The circulation time was 20 seconds. Operation was done March 6, 1937. The patient states that he has felt better since the operation and is able to make fifty-two trips on the steps, stopping because of moderate dyspnea. He works in his garden and does much physical work about the house, without symptoms. He has been unable to find work.

CASE 24.—Arteriosclerotic heart disease and angina pectoris of five years' duration. A tailor, aged 50 years, in November, 1933, and again in August, 1936, had myocardial infarcts. They were anterior and apical, and posterior and basal. Invalidism followed the second attack and he was barely able to go to his shop. Slight exertion and emotional strain brought on pain. The left ventricle was slightly enlarged. The circulation time was 23 seconds. The venous pressure was 15 cm. of physiologic solution of sodium chloride. The patient had extensive myocardial fibrosis and was a doubtful candidate for operation. Operation was done April 9, 1937. At the operation the heartbeat was feeble, and it was apparent that we were dealing with a seriously damaged muscle. The patient stated that he had been greatly relieved from pain following the operation. That the patient went through the operation and received some relief from pain seems to be significant, because we believe that he had a damaged myocardium. He continued to have some pain on emotional and physical strain, and his exercise tolerance as evidenced by the two-step test was not increased. He died one year and two months later of myocardial infarction. A post-mortem examination was obtained.

CASE 25.—Arteriosclerotic heart disease and angina pectoris of nine years' duration. A retired salesman, aged 52 years, had had some attacks which were sufficiently severe to have been caused by coronary thrombosis. The conventional electrocardiogram was normal, but the T wave in the chest lead was reversed in direction. The record taken during the induced anginal attacks after exercise showed changes suggesting a posterior and basal infarct. Operation was done April 24, 1937. The patient states that there is improvement and he is able to carry on his work, without symptoms.

CASE 26.—Arteriosclerotic heart disease and angina pectoris for one and one-half years; an attacks of myocardial infarction one year before operation. A female elementary school principal, aged 50 years, had severe angina of effort and of emotion which totally incapacitated her. She was able to make but three two-step trips, stopping because of typical angina. Operation was performed on July 30, 1937. Her convalescence was slow, but on Oct. 11, 1937, she was able to make twelve trips, and on Nov. 29, 1937, this was increased to twenty. In the intervening period, the patient has been able to walk a few blocks slowly, without pain, but is unable to return to work.

CASE 27.—Arteriosclerotic heart disease and angina pectoris following an attack of coronary thrombosis two years previously. A factory superintendent, aged 37 years, had angina of effort which caused him much distress. He was operated upon on Aug. 4, 1937, and his convalescence was uneventful. He left the hospital in thirty-seven days. He was able to return to his job, working five to eight hours a day. He still has occasional pain for which he takes nitroglycerin.

CASE 28.—Arteriosclerotic heart disease and angina pectoris of six years' duration. A salesman, aged 50 years, was unable to carry on with his work, and he was operated upon on Aug. 13, 1937, making an uneventful convalescence. He gradually improved and finally was able to do moderate exercise without any pain. He has steadily improved.

POST-MORTEM EXAMINATION OF HEARTS OF PATIENTS WHO RECOVERED FROM THE OPERATION BUT WHO DIED LATER

CASE 3.—The specimen was studied three days after death and after embalming. The grafts were everywhere atrophic. All large coronary arteries showed severe sclerosis. Barium-gelatin mixture injected into the two coronary arteries did not inject into the entire arterial bed very satisfactorily. None of the injected mass flowed forth into the grafts. It was concluded that if there were any in the grafts, they were not large enough or numerous enough to carry any significant quantity of blood. However, the injection of the coronary bed was not satisfactory.

CASE 20.—The coronary arteries showed a severe degree of sclerosis. Injection of these vessels with barium-gelatin mixture was carried out. This injection mass was of a consistency thicker than blood. It ran away from the grafts in rapid drops, but not in a constant stream. When the specimen was cleared, ten or twelve communications could be observed between the arterial system and the grafts. These vessels were large enough to carry blood. The specimen showed vessels that could have explained the clinical improvement.

CASE 24.—The coronary arteries showed severe sclerosis. They were injected with barium-gelatin mixture. A few small vessels connected the arterial system with the vessels of the grafts. These were not numerous enough, and they did not appear to be large enough to carry any significant quantity of blood.

SUMMARY

Of the thirty patients operated upon, thirteen (65 per cent of those surviving) were definitely improved. Their clinical symptoms were either completely or almost entirely relieved, so that they could resume their previous occupations. Four patients (20 per cent of those surviving) were moderately improved, while in three instances (15 per cent of those surviving), there was inconsequential improvement. An anatomic study of three specimens was carried out. In one of these, the

clinical result had been good and the specimen showed vascular anastomoses. In this group of twenty surviving patients, the high percentage of patients showing great improvement is not only significant but encouraging. This statement may be emphasized because all of these patients were seriously disabled before the operation.

ADDENDUM

In the year since this report was made, there have been no deaths and the patients have all shown improvement. Case 9 has worked steadily as a painter and has had no pain. Case 17 likewise has worked steadily as a mechanic—doing hard work. He has had no pain and continues his thyroid extract for myxedema. Case 26 developed a depression and was treated by electrically induced convulsive seizures. This treatment was borne with no untoward effects. She has retrosternal pain occasionally on effort but feels greatly improved.

During the past year measurements were made in our surgical research laboratory concerning the intercoronary communications (Stanton, Schildt, and Beck). It was known from our early experimental work that vascular communications between coronary arteries could be produced by operative methods. The branches of one coronary artery can be made to communicate freely with branches of other coronary arteries. Various surgical methods are available to produce a common coronary bed. We used abrasion of the surface of the heart as one method to accomplish this purpose. We also placed powdered beef bone on the abraded surface of the heart to produce a foreign body reaction. A common arterial bed protects the heart in the presence of coronary artery occlusion. It is possible to ligate an important coronary artery, such as the descending ramus of the left at its origin with a reduction in mortality and with a diminution in the size of the infarct that develops after occlusion of the artery. Indeed, it is possible to ligate a major coronary artery without having any infarct develop subsequently. This is never possible in a normal heart.

We believe that the work has advanced to the place where preliminary or limited application to human patients should be done. We have waited over three years since the last human patient was operated upon in order to obtain further data on the subject. Satisfactory data have now been accumulated. The experimental evidence is good. The improvement in some of the patients has been almost incredible. We do not feel that general application should be carried out at present, but we do feel that we should go ahead in the immediate future with another preliminary group for study.

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DISCUSSION

DR. JOHN W. STRIEDER, Boston.—I merely wish to place in evidence a bit of pathologic information because Dr. Beck asked me yesterday to do so.

This patient, upon whom we had performed an omental graft after the method of the late Mr. O'Shaugnessy, was 45 years of age. He was a salesman who had been observed by Dr. Graybiel, my colleague, for some time. He had unequivocal evidence of angina, and his exercise tolerance as manifested by the two-step test was extremely low. He could not walk across the street without inducing an attack. He withstood cardio-omentopexy well, and during the ensuing six months had marked improvement, so that he was able to return to his job without medication and without much pain. He had some pain but it took a great deal of effort to induce it. He boasted to his friends of his "new heart."

About six months after his original operation, he began to have gastric symptoms, and shortly thereafter it became evident that he had an acute abdominal emergency, with the stomach partly incarcerated in the hiatus created in the diaphragm. This was closed without difficulty. The patient stood the operation for the repair of the diaphragmatic hernia well and was returned to his bed in good condition. He died suddenly forty-five minutes later, apparently from a cardiac episode.

At autopsy, the graft was securely in place, and on injecting the specimen through the superior mesentery artery, it was evident on section and on x-ray that there was a rather rich vascular ingrowth from the graft into the myocardium. There was diffuse fibrosis throughout the myocardium.

I mention this case as a correlation of pathologic evidence with clinical improvement.

CINEROENTGENOGRAPHIC STUDIES OF THE PULMONARY CIRCULATION, THE CHAMBERS OF THE HEART, AND THE GREAT BLOOD VESSELS IN HEALTH AND DISEASE

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RECENT achievements in roentgenology have added greatly to the accuracy of diagnosis of intrathoracic disease. The ease of detection of even small lesions within the parenchyma of an aerated lung is a matter of common knowledge. In the case of the cardiovascular system, however, roentgenologic diagnosis depended on changes in the gross outline of the structures. Exact diagnosis on the basis of the radiographic findings alone was often impossible because of lack of contrasting densities. The recently described method of Robb and Steinberg[1] for the visualization of the great vessels, cardiac chambers, and pulmonary vessels was a great advance in the roentgenology of the cardiovascular system. By the rapid intravenous injection of a radiopaque substance, the large intrathoracic vascular structures are delineated, and abnormalities may be diagnosed with remarkable accuracy. As the contrast medium passes rapidly through the heart and great vessels, only a few seconds are available for the recording of the opacified structures on a roentgen film. Without special equipment, only two roentgenograms can be made in the brief time before the contrast medium passes from the aorta. Consequently some of the structures may not be depicted because of absence of opacification of a particular vessel at the moment the roentgenograms are made. The application of cinefluorography,[2] in combination with the visualization method of Robb and Steinberg, permits the recording of the passage of the opaque blood through the subclavian and innominate vein and superior vena cava into the right auricle and ventricle, through the pulmonary vessels, back into the left auricle and ventricle, and finally into the aorta.

Employing the method described by Robb and Steinberg, 30 to 40 c.c. of 70 per cent diotrast is injected rapidly into a large arm vein. The passage of the opacified blood through the heart and great vessels, as seen on a special fluoroscopic screen, is recorded by a motion picture camera. In cases in which the circulation time is normal, or nearly so, the course of the contrast medium may be followed from the vena cava

From Lenox Hill Hospital, New York, N. Y.
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to the aorta with a single injection. If studies are desired in more than one position, additional injections have to be made.

The success of visualization of the great vessels and cardiac chambers by diotrast depends on the rapidity of the intravenous injection, so that the amount of mixing with the blood is reduced to a minimum. As the opaque medium passes through the heart and pulmonary circuit, more and more mixing with blood occurs, and, therefore, the opacification is less marked. If the cardiac chambers are dilated, the diotrast becomes so diluted within the heart that the aorta, or even the cardiac chambers, are not visualized. Dilation of the aorta also lessens the sharpness of contrast.

Cineroentgenographic studies of the heart and great vessels during visualization of these structures with diotrast has demonstrated a number of pathologic conditions, including the following:

1. Disturbance of blood flow through the great veins.

2. The actual size of the pulmonary artery, with its variations and certain deformities, such as aneurysmal dilatation and constriction.

3. The cardiac chambers with demonstration of the actual thickness of the ventricular walls, and also the abnormal contracture of the wall of a cardiac aneurysm.

4. The demonstration of aneurysm of the great blood vessels, as well as deformities, angulations, and displacements of the circulatory system in the thorax, caused by pathologic processes.

5. The visualization of some of the types of defects present in congenital heart lesions.

6. The demonstration of a defect in the septum between the right and left cardiac chambers.

Recently we have been particularly interested in the investigation of cases of congenital cardiac disease by this method. Until the present time only a small number of these cases have been studied. It was found that by the use of the cineroentgenographic method, certain abnormalities could be better demonstrated than when roentgenograms only were made, because the entire cardiac cycle was visualized in the former method. This enabled the detection of such lesions as septal defects in the heart. Certain factors of importance which will influence the possibility of demonstration of such abnormalities may be briefly discussed.

In the visualization of a shunt between the right and left side of the heart or between the pulmonary artery and aorta, the direction of blood flow in the shunt is of considerable importance. In the cyanotic group of congenital cardiac cases, the demonstration of a shunt will be easiest because in these cases the blood flow through the shunt is from right to left. Therefore, if diotrast has been injected, the blood is still well opacified as it passes through the shunt. If, however, the blood flow through an intraventricular septal defect, for example, is from left to

right, much more dilution of the opaque medium would have occurred before the opacified blood passed through the defect. As a result, it might be difficult or impossible with present methods to demonstrate the septal defect. It would be difficult to demonstrate blood flow from the aorta through an uncomplicated patent ductus arteriosus into the pulmonary artery. If other anomalies were associated with the patent ductus arteriosus, however, and the blood flow through the ductus was from pulmonary artery to aorta, the shunt would be more readily demonstrable. It is questionable whether the ductus arteriosus itself could be visualized because of its short length and superimposition on the shadow of the pulmonary artery and aorta in the film. The presence of the patent ductus might be demonstrated indirectly, that is, by demonstrating the passage of blood from the pulmonary artery into the aorta.

Cineroentgenography combined with the injection method of Robb and Steinberg may be able to clarify our knowledge of the living anatomy and physiology of the central portion of the circulatory system. The demonstration of pathologic conditions has been discussed. Further technical improvements may considerably enlarge the field of usefulness of cineroentgenography.

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A MECHANISM FOR ARTIFICIAL PULMONARY VENTILATION IN THE OPERATING ROOM

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OUR interest in this problem is an outgrowth of experimental and clinical problems in the field of cardiac surgery.[1, 2] As a result of our attempts to revive the human heart from ventricular fibrillation, we became aware of the need for a means of carrying out artificial pulmonary ventilation in the operating room over relatively long periods of time as a preliminary to successful resuscitation. We first constructed for use in the operating room a type of respirator for intermittently insufflating compressed air down a tracheal catheter, such as is commonly used in physiologic laboratories. With such a respirator available, we found several opportunities to use it in respiratory failure from other causes. The greatest degree of success was in a patient with complete respiratory failure caused by increased intracranial pressure. The mechanical respirator was used for several hours while Dr. C. S. Beck removed a brain tumor, following which the patient made an uneventful recovery and lived a normal life for two years. During the past four years there have been several other instances in which it seems reasonably certain that fatalities have been averted in the operating room by the use of this respirator.

We found several disadvantages to the previously mentioned respirator. It is an open method and requires the insertion of a tracheal catheter, which is time-consuming. Consequently we worked with the idea of having a mechanical respirator as an integral part of the commonly used closed system gas anesthesia machines. This was accomplished by inclosing the breathing bag of the anesthesia machine in a rigid container and having a mechanism for intermittently injecting compressed air between the container and the bag. This applies intermittent pressure to the gas in the bag and mechanically propels gas from the bag into the lung, the elastic recoil of the thorax and lung being sufficient to return the exhaled gases. Suction during the expiratory phase, as has been advocated in some "resuscitators," seems neither necessary nor desirable.

It may be necessary at this point to establish the advantages, if any, of a mechanical method over the commonly used method of compressing the breathing bag by hand. The advantages appear to be (1) the ability to standardize precisely the bag pressure fluctuations as to rate and time-pressure relationship, and (2) the ease with which such a method can be

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employed over long periods of time. Such a method is not intended to replace all other methods of artificial respiration, but rather to supplement these methods.

Fig. 1.—Photograph of mechanical respirator attached to kinetometer of Heidbrink anesthesia machine. The respirator is operated by compressed air under pressure of 4 pounds per square inch. Rate and peak pressure are regulated by the two controls on the top of the respirator.

After our preliminary experiences with such a respirator,[3, 4] Mr. O. J. Smith of the Ohio Chemical and Manufacturing Company has undertaken the work of perfecting the mechanism of this respirator and has built the unit shown in Fig. 1. A very important part of the mechanism is a pressure-regulating device which determines the maximum pressure which will be permitted to develop around the breathing bag. By varying (1) the rate of inflow of compressed air, (2) the rate of alternations

of the escape valve, and (3) the peak pressure, a great variety of pressure-time relationships can be obtained (Fig. 2). The principle appears to be identical to that used in a similar apparatus constructed in the clinic of Crafoord of Stockholm, Sweden, and used by him in open chest operations.[5, 6]

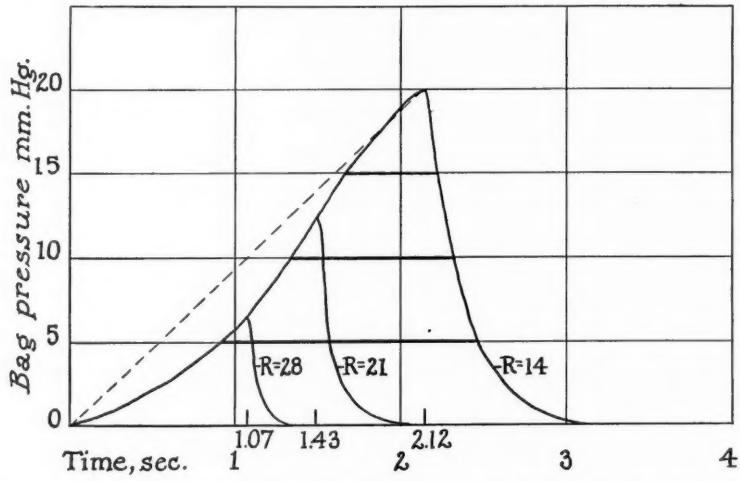

Fig. 2.—Variation of time-pressure relationship possible by varying (1) rate and (2) peak pressures.

Our mechanism was designed for use in cases of advanced respiratory failure. If it could be shown that lesser degrees of respiratory insufficiency during operation produce significant body disturbance, there might be indications for more extensive use of artificial pulmonary ventilation. Studies in the pathologic physiology of open pneumothorax suggest that the constant flow positive pressure methods fall short of maintaining a normal pulmonary ventilation. In both the laboratory and the operating room it can be observed consistently that following open pneumothorax there is *increased* respiratory effort and *diminished* minute volume of respiration regardless of what pressures are used for the inspired gas mixture. This increased respiratory effort may gradually increase for the duration of the open pneumothorax. Such observations are more striking in the laboratory because of the delicate mediastinum of the dog. Since a bilateral open pneumothorax is not very frequently produced in man during thoracic operations, intrathoracic operations on man do not disturb pulmonary ventilation so frequently and so severely as in our experimental animals. However, the surgeon must be prepared for every eventuality.

The increased respiratory effort following open pneumothorax suggests an accumulation of carbon dioxide in the blood. Lenhart,[7] in 1920, reported studies showing that in open pneumothorax in rabbits without

positive pressure respiration there was: (1) Marked increase in respiratory effort and rate; (2) a decrease in minute-volume of respiration; (3) impairment of the elimination of carbon dioxide before oxygen uptake was reduced, leading to acidosis due to carbonic acid, with decrease in the hydrogen-ion concentration of the blood.

Crafoord found in dogs that with constant flow positive pressure there was a gradual accumulation of carbon dioxide in the organism during the time that there was yet no decrease in oxygen content of the arterial blood. We have repeated some of these studies to compare (1) constant flow positive pressure and (2) intermittent positive pressure.

Bilateral open pneumothorax in the dog or in man will prove rapidly fatal unless there is enough pressure in the lung to enable at least parts of the lung to follow the movements of the chest wall. A number of experiments were done on dogs to study pulmonary ventilation in open pneumothorax under various conditions.

EXPERIMENTAL PROCEDURE

Dogs were anesthetized with intravenous barbital. No morphine was given in order to avoid depression of the respiratory center. Barbital was used because of its prolonged action, in order that the response of the respiratory center should not vary as a result of varying depth of anesthesia. A tracheal cannula was tied in place and connected to a Heidbrink constant flow anesthesia machine. Pure oxygen was administered and carbon dioxide absorbed from the expired air. The pressure fluctuations of the lung-bag system were constantly observed with either a water or mercury manometer. Arterial blood samples were drawn under oil from the femoral artery. Carbon dioxide determinations were done in duplicate on the Van Slyke apparatus. Hematocrit determinations were made with Wintrobe tubes.

COMMENT ON EXPERIMENTS

With a constant level of anesthesia, there was a very constant correlation between carbon dioxide content of the arterial blood and the respiratory effort. In all, ten such experiments were performed, and Fig. 3 is a graphic representation of a typical experiment. The initial carbon dioxide determinations were done thirty minutes after administration of the anesthetic and connection of the animal to the anesthesia machine. Open pneumothorax was produced, and the flow of oxygen was regulated so as to maintain the desired pressure level in the lung-bag system, which in this instance was 10 mm. Hg at the end of expiration. The mechanism of pulmonary ventilation under these conditions is as follows:

The lung in contact with the chest wall is allowed to *expand* during *inspiration*, increasing the volume of the lung-bag system and lowering the pressure, in this case to 6 mm. Hg. A fluctuating pressure such as this will be described by giving expiratory and inspiratory values as 10/6 mm. Hg. As the intrapulmonary pressure is lowered, areas of the lung surrounded only by air at atmospheric pressure *contract* during *inspiration*. There is this paradoxical excursion of different parts of the lung. This can perhaps be illustrated best in the human being with separate pleural spaces. Fig. 4 indicates the inspiratory and expiratory positions of the thorax in open pneumothorax at a hypothetical pressure of 6/4 mm. Hg. This pendulum movement of the air from one lung to the other, as pointed out by Crafoord, as a result of the paradoxical excursion is equivalent to increasing the dead space of the respiratory tract.

With the constant flow positive pressure, the carbon dioxide content of the arterial blood increased about 10 volume per cent in two hours. This has been consistently noted in all experiments, but the rise was not so great as noted by

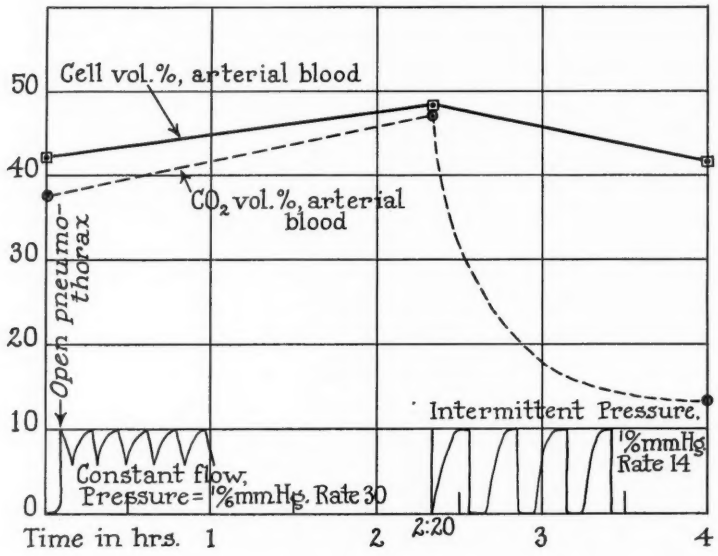

Fig. 3.—Comparison of constant flow positive pressure and intermittent pressure of equal magnitude in open pneumothorax in the dog.

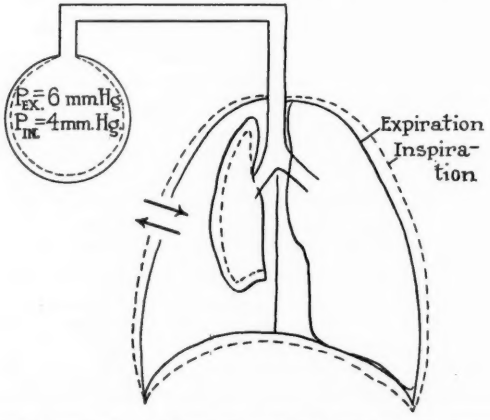

Fig. 4.—Paradoxical excursion of partially collapsed lung in open pneumothorax with constant flow positive pressure respiration.

Crafoord, possibly because he used slightly lower pressures. In no instance have we seen the blood as effectively cleared of carbon dioxide with this method of ventilation as with the chest intact. After two hours, intermittent pressure was started with the respirator, using pressure of 10/0 mm. Hg in order to have the

same peak pressure as with the constant flow method. In spite of the fact that the peak pressure was the same and the rate was only one-half that of the spontaneous breathing, the animal was being hyperventilated as indicated by the rapid onset of apnea and the low carbon dioxide content of the arterial blood after one hour and forty minutes. The hematocrit reading showed considerable hemoconcentration during the accumulation of carbon dioxide, with blood dilution occurring after removal of carbon dioxide. This finding has been consistently found and agrees with the studies of Van Slyke, Wu, and McLean[8] on carbon dioxide retention. This hemoconcentration and shift in the hydrogen-ion concentration of the blood are factors unfavorable to a patient's well-being that may readily be overlooked and no doubt are worthy of more study in the anesthetized patient.

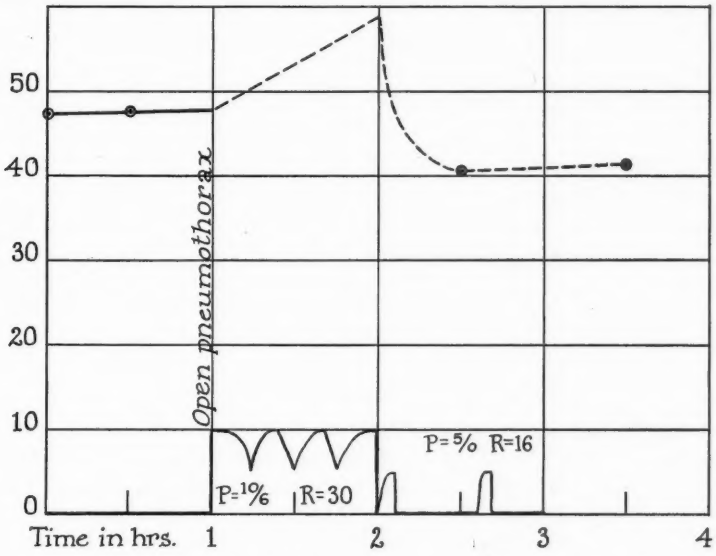

Fig. 5.—Comparison of normal breathing, open pneumothorax with constant flow positive pressure of 10/6 mm. Hg, and open pneumothorax with intermittent positive pressure of 5/0 mm. Hg in the dog.

As was pointed out, pressure 10/6 mm. Hg was far inferior to 10/0 as far as pulmonary ventilation is concerned. As we decrease the pressure in the first type of respiration, the ventilation becomes progressively poorer until at pressure over 4/3 a fatal outcome was noted after three to four hours in two instances. However, it was found that with intermittent pressure 5/0 we obtained better pulmonary ventilation than with the chest intact (Fig. 5).

CLINICAL APPLICATION

The experiments leave no doubt that in open pneumothorax intermittent pressure gives better pulmonary ventilation than constant flow positive pressure. It makes no difference how we produce this pressure, but we should think in terms of restoring respiratory physiology to as near normal as possible during intrathoracic operations. The surgeon need have no fear of bilateral open pneumothorax, since with intermittent pulmonary pressure normal pulmonary function can be maintained

indefinitely. We have used the mechanism herein described in about twenty-five operations upon the heart and lungs and have found the information from the laboratory can be applied directly in the operating room.

Intermittent pressure 5/0 mm. to 10/0 mm. Hg in open pneumothorax clears the blood of carbon dioxide sufficiently well to keep respiratory effort as quiet as in the resting, unanesthetized person with the chest intact. There seems no necessity, except possibly in unusual circumstances, to hyperventilate a patient to the extent that respiratory effort entirely ceases.

Such a mechanical respirator used on a patient with an intact chest requires higher pressures to produce an adequate minute volume of respiration, since the chest must be lifted and the diaphragm pushed down. Consequently, the use in such a situation is indicated only when there is definite respiratory insufficiency. Such a mechanism should not be used to hyperventilate a patient when the physiologic mechanism for doing this is intact. It is recognized that many of the details regarding the scope of clinical application of this mechanism need to be worked out and defined more clearly. So far, an attempt has been made to establish the broad physiologic principles for the application of an intermittent pressure mechanism in intrathoracic operations.

SUMMARY

A mechanism has been described for intermittent pulmonary insufflation in the presence of open pneumothorax. It has been demonstrated that an equal degree of pulmonary ventilation can be obtained with *lower* intermittent pressure than with a constant flow of positive pressure. Such a mechanism is suggested for use in the operating room in case of respiratory insufficiency from other causes.

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COMPLICATIONS OF EXTRAPLEURAL PNEUMOTHORAX

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EXTRAPLEURAL pneumothorax has been used at the Westfield State Sanatorium for the past eighteen months in the treatment of pulmonary tuberculosis. While we have had satisfactory results in many cases, we have seen various complications, some of which have not been reported in the literature. It is the purpose of this paper to give our impressions of these complications, and the methods we have used in handling them.

As others have stated, we believe that extrapleural pneumothorax has a low immediate operative mortality and that it can be used on extremely ill patients, but also that it has many potential late complications and, therefore, in the long run, is far more hazardous than thoracoplasty.

The complications we have listed as follows: (1) re-expansion of the lung before healing occurs; (2) spread of disease; (3) air embolism; (4) hemorrhage into the extrapleural space; (5) acute intestinal tuberculosis; (6) tuberculous infection of the extrapleural space; (7) bronchoextrapleural fistulas; and (8) submuscular abscess.

Some of these complications are attendant upon any operation in the presence of tuberculosis. Others are complications of the operation itself. Adequate collapse was obtained in all cases immediately postoperatively. However, partial re-expansion with resultant inadequate collapse occurred in seven of our cases. One occurred within a month after operation because of uncontrollable violent cough. This, we have found, is an important cause of re-expansion. In two cases, it occurred at two months; two also occurred at five months after operation one at six months, and one at eight months. All of these patients were kept at positive pressure readings, with refills at short intervals.

Contralateral spread of the disease took place immediately after operation in one case and this has subsequently cleared. It was found twenty-five days after operation in another case, and now ten months postoperatively this has cleared. Another was found to have spread three months after operation, and one case each at four, six, and thirteen months.

We have had one case of what we believe to have been air embolism occurring three days after operation. It was not fatal, although the

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patient was critically ill for six hours. There is a real as well as a theoretical danger in the use of positive pressure in the extrapleural space.

We have had one hemorrhage into the extrapleural space which occurred in the first week after operation. There was more bleeding than usual at the time of operation, but this we thought had been controlled adequately. This blood was removed and replaced by air. There was not sufficient blood loss to require transfusion, and the patient is now continuing his refills.

There have been two cases of acute intestinal tuberculosis. In one of these, it appeared six weeks postoperatively and was accompanied by nutritional edema. These complications caused the patient's death two months after operation. The sputum was negative at this time, and the pulmonary cavity was found completely collapsed by the extrapleural pneumothorax at post-mortem examination. The other patient developed symptoms of intestinal tuberculosis three months after operation. Since the institution of pneumothorax on the contralateral side, followed later by intrapleural pneumolysis, the intestinal symptoms have subsided and the patient's general condition is greatly improved.

We have had seven patients in whom the fluid in the extrapleural space has been found to contain tubercle bacilli, either on smear or by guinea pig inoculation. One was positive on smear at three months and developed a bronchoextrapleural fistula at four and one-half months. One was proved by smear at four months; one was found by guinea pig inoculation at six months, and two were shown by smear at ten months. One also was proved by guinea pig inoculation eleven months after operation. One case easily held with a good collapse developed fluid at fifteen months which was found positive by smear. As all of these were found at three months or longer after operation, we believe that thoracoplasty should be done to close this space as soon after the extrapleural pneumothorax as the patient's condition warrants.

We have had four bronchoextrapleural fistulas. One occurred twenty-five days after operation with a rupture of the lung and inundation of the contralateral lung, followed by the development of extensive disease or pneumonitis. This shows the advisability of removing excessive fluid from the extrapleural space and not allowing patients to sleep on the good side. X-rays at present show marked clearing of this contralateral disease in the case described, and the patient is gaining weight. He will have a thoracoplasty over this space as soon as his condition permits. Another patient in whom the parietal pleura was opened and the interlobar fissure entered during the operation developed a bronchoextrapleural fistula one month after operation. She has since had a thoracoplasty over this space. Another similar case in which the interlobar fissure was entered developed bronchoextrapleural fistula two months postoperatively. The fourth case is one previously referred to

in which the patient developed fluid which was positive for tubercle bacilli on smear at three months. Refills were abandoned to allow the lung to expand in preparation for thoracoplasty, and as a result, a fistula occurred at four and one-half months. Evidently the cavity ruptured from too high a negative pressure in the extrapleural space. We now proceed with thoracoplasty over the extrapleural space without waiting for re-expansion, and remove air from the space on the operating table after anesthesia has been induced.

We have drained externally one of these extrapleural spaces with a bronchoextrapleural fistula before doing a thoracoplasty. We do not believe that we should do this again unless there was a large collection of fluid in the extrapleural space which could not be removed by aspiration. So far, we have not found this large an amount of fluid present in the cases with a fistula. In fact, our first suspicion of a fistula is the rapid disappearance of fluid from the space and a reading at atmospheric pressure. We confirm the presence of fistula by injecting Congo red into the space, with recovery in the sputum.

When a bronchopleural fistula develops in the presence of extrapleural pneumothorax, we prefer to treat the space as a large pulmonary cavity rather than a mixed infection empyema with a bronchopleural fistula, and we feel that thoracoplasty is indicated. We operate upon such patients in the Trendelenburg position and use suction with a catheter to remove secretions which may drain while under anesthesia.

It is our belief that extrapleural pneumothorax in most cases should represent the first stage of an ultimate thoracoplasty. Thoracoplasty should be done as soon as the patient's general condition warrants, so that this potentially hazardous space may be obliterated.

It has been our experience that patients tolerate thoracoplasty over this space remarkably well. In the eight thoracoplasties over an extrapleural pneumothorax which we have done to date, we have removed from four to seven ribs at one operation. In our first cases we were more conservative as to the number of ribs removed. As stated above, it has been our policy in these cases without fistulas to aspirate the air on the operating table after anesthesia has been induced and before the operation has begun. Subsequent postoperative aspirations of air during the first week have been done under the fluoroscope so that the space may be obliterated and the regenerating rib will hold the space collapsed. We do not believe that an extrafascial apicolysis is necessary or safe, because in these cases the apex of the lung is usually below the third or fourth rib. It would be a particularly hazardous procedure in the presence of a tuberculous or mixed infection within the extrapleural space. We do not resect the transverse processes above the third.

The present status of thoracoplasties we have performed and the reasons for doing each are listed in Table I.

TABLE I

NAME	INTERVAL BETWEEN EXTRA-PLEURAL PNEUMO-THORAX AND THORACO-PLASTY	REASONS FOR THORACOPLASTY	DATES OF THORA-COPLASTY	STAGES	NO. OF RIBS	PRESENT STATUS
P. C.	13 mo.	Cough; sputum; bronchoextra-pleural fistula	8/25/39 9/26/39	2	4 } 8 4 }	Sputum positive; cavity on other side; cough and sputum diminished; eating well for first time since admission
E. H.	1 yr.	Fluid positive by guinea pig inoculation Aug. 8, 1939, although sputum negative	10/ 3/39	1	5	Complete obliteration of space; closure of cavity; negative sputum
M. M.	9 mo.	Bronchoextra-pleural fistula	4/27/39	1	7	Submuscular perforation of space, spreading disease; poor result
E. N.	9 mo.	Re-expansion of lung; positive sputum; fluid in space positive by guinea pig inoculation	5/16/39 6/13/39	2	5 } 7 2 }	Negative sputum; space obliterated
S. B.	9 mo.	Partial re-expansion of lung; operated upon before pulmonary lesion reactivated	5/23/39 6/27/39	2	4 } 7 3 }	Extrapleural space obliterated; sputum negative to smear and by guinea pig inoculation; gaining weight
J. C.	7½ mo.	Re-expansion of lung, with recurrence of positive sputum; space fluid positive on smear June 4, 1939	8/15/39 9/ 5/39	2	4 } 8 4 }	Space obliterated; sputum negative
D. B.	8 mo.	Re-expansion of lung; cavity re-opened; sputum positive	2/14/39 6/13/39	2	5 } 8 3 }	No cough or sputum; negative sputum; gaining weight
C. B.	5 mo.	Huge peripheral cavity never closed; broncho-extrapleural fistula	4/21/39 6/27/39	2	3 } 3 0 }	Took anesthesia poorly; glands found in gutter at first stage; submuscular abscess found at second stage; respiratory reserve poor

Another complication we have encountered was a submuscular abscess connecting with the extrapleural space; this was found at thoracoplasty in one case. It was caused by a rupture of the posterior end of the periosteal bed through which the original operation had been done. Another patient was found to have a tuberculous abscess below the muscles when the incision was reopened for the second stage of the thoracoplasty. At the first stage lymph nodes were found in the area near the spine and ribs. These were evidently the cause of the abscesses found at the second stage. Glands similar to these have been seen at the first stage of an uncomplicated thoracoplasty and have shown tubercles microscopically. As yet, we have not found such abscesses at a subsequent stage of a thoracoplasty. This complication is a serious one because, as shown by the data of Table I, these two patients are the only ones who have not done reasonably well. It may be that poor resistance to their disease is the underlying factor.

It is interesting to note that in all the patients developing a bronchoextrapleural fistula, the operation of extrapleural pneumothorax was difficult. The layer between the parietal pleura and the endothoracic fascia was difficult to develop in many places. With resection of a portion of the fourth rib posteriorly, we are, in most cases, directly over or very near the interlobar fissure between the upper and lower lobes of the lung. We have resected the fifth rib in a few cases and have found that the identification of the correct layer was much easier and that the apex of the thoracic cavity was as easily reached as through the periosteum of the fourth rib. Entering the parietal pleural at the interlobar fissure is dangerous, because in the two cases of this group in which it occurred bronchoextrapleural fistulas developed.

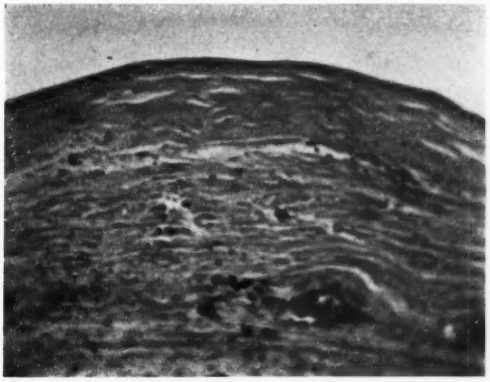

Fig. 1.—Case D. W. Section from lining of the extrapleural pneumothorax space.

Careful closure of the periosteal bed of the resected rib should be done. Inadequate closure will allow the escape of air, which is necessary to maintain this space. It also will allow the leakage of fluid which

forms immediately after operation, and if this fluid becomes infected with tubercle bacilli, a submuscular abscess may form.

We have made post-mortem examinations in the fatal cases, one at two, and the other at six, months after operation. It is interesting to see a microscopic section of this tissue which lines the extrapleural space. It is scar tissue which, like all scar tissue, has a poor blood supply. According to our sections, we do not find a mesothelial lining which others have described as forming the lining.[1]

In cases with disease of long duration, we believe that the operation of extrapleural pneumothorax will be more difficult and more hazardous than in those of shorter duration. In these cases the tissue overlying the cavity may derive some of its blood supply through the endothoracic fascia, so that separation of the parietal pleura may interfere with the blood supply and allow rupture of the cavities by necrosis.

All of the chest fluids in these cases were examined routinely by smear, concentration, and guinea pig inoculations, as were the sputum specimens.

SUMMARY

We have listed the complications which have presented themselves in a series of twenty-one cases of extrapleural pneumothorax. Some of these are complications of the operation itself, and others are complications of any operation on a tuberculous patient. We find that the extrapleural pneumothorax is attended by a low immediate postoperative mortality rate—we have had no immediate mortality in twenty-one cases—but we believe that its potential complications are far more serious than those of extrapleural thoracoplasty. We believe that the potentially hazardous extrapleural space should be obliterated by thoracoplasty as soon as the patient's general condition permits.

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PRIMARY MULTIPLE ALVEOLAR CELL TUMOR OF THE HUMAN LUNG

Report of a Case

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THE existence of true, primary, multiple, malignant tumors arising in the pulmonary alveoli is still questioned by many observers. Ewing, in the 1940 edition of his book on neoplastic diseases, gave a review of the older literature which we, therefore, omit; he described, besides a diffuse form, a multiple and nodular form of carcinoma arising from the pulmonary alveoli. Weissmann reported two cases of the latter form and believed the tumor to be based upon an embryonic malformation of the alveolar epithelium, which in an individual with general predisposition to tumors may be transformed into cancerous tissue. In his observations the alveoli were lined with cuboidal or cylindrical tumor cells, showing in places papillomatous protrusions into the lumen. Similar cases were reported by Reuss. Also Oberndorfer[a] advocated the existence of an "alveolar cell cancer," on the grounds of his personal experience with several cases. Other writers, however, were of the opinion that no proof exists of the origin of lung cancer in the pulmonary alveoli. Frissell and Knox observed one case of "bilateral miliary carcinoma" in forty-six pulmonary cancer patients examined. The gross aspect of the tumor was like that of metastases, with evenly distributed nodes in both lungs. The authors believed that the cancer arose "from some small area which could not be distinguished from the mass of miliary infiltration." They pointed out that pulmonary tumors are essentially bronchogenic in origin* and called attention to the fact that bronchogenic adenocarcinoma has "a tendency to replace the lining cells of the alveoli." Lindberg said that there is little likelihood of a primary multicentric pulmonary cancer with alveolar epithelium as the starting point; he believed that all such cancers are bronchogenic in origin. D'Aunoy, Pearson, and Halpert stated that they had never seen origination of cancer from the alveolar lining cells. Recently, Casilli and White described a case of

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*Fabris advocated the opposite view; he said that the alveolar cell is the starting point for the majority of lung tumors; however, he did not describe neoplasms comparable to those discussed in the present paper.

what they named "carcinomatoides alveogenica multicentrica." They did not find a primary tumor elsewhere in the body; the bronchial tree, too, was free from tumor. The growth was multicentric, affecting all lobes; histologically, it was confined to the alveolar sacs, sparing the interstitial framework; and it was unlike any other primary lung cancer, the air sacs being filled with polygonal neoplastic cells. All these facts led the authors to the conclusion that the tumor sprang from the alveolar sacs. The new designation was chosen because of the uncertainty as to the nature of the alveolar lining cells.

Diffuse tumors, possibly arising in the pulmonary alveoli, seem to be still rarer than nodular tumors. Sweany published a report of "a so-called alveolar cell cancer of the lung" in a 47-year-old male. The right lung and the middle portion of the left were a "consolidated mass of ashen gray appearance. The cells first supplanted the normal alveoli and maintained an alveolar arrangement. Later they formed papillary growth into the alveoli. . . . The origin seems probably to have been somewhere between the lower respiratory bronchioles and the alveoli." According to Sweany, there is no proof as yet that the alveolar cells are the parent tissue. Scattered smaller nodes in the left lung were considered to be metastases. Bonne reported a case of a 30-year-old Chinese male in whom the bulk of the lung parenchyma was replaced by a diffusely growing homogeneous tumor, resembling hepatization. He called this condition carcinosis. Metastasis was not found. The microscopic features resembled those described by Weissmann. Mitosis was frequent. The alveolar septa were preserved. The terminal bronchioles would be involved in a similar manner. The alveoli and respiratory bronchioles represented the very seat of the disease. Morphologically, the carcinosis resembled closely certain lung diseases of animals, especially the so-called "jagziekte" in sheep.*

REPORT OF A CASE

The following case affords an opportunity to elucidate the pathogenesis and clinical diagnosis of the nodular tumors under discussion.

M. Q., a 39-year-old WPA worker who had been a miner from 1918 to 1936 and whose previous history was not remarkable, began to cough a little during August, 1938. The cough soon became severe and more persistent, and the patient lost weight. He felt weak and had epigastric distress following meals. When he entered the Colorado General Hospital (Nov. 17, 1938), he appeared poorly nourished. The temperature was normal; respirations were 24; and blood pressure was 86/58. Breath sounds were slightly harsh in both apices. Bases were clear. There was generalized epigastric tenderness, but gastric function tests were essentially negative. Other laboratory tests disclosed nothing of significance. X-ray examination showed gastroenteroptosis, and both lungs were studded with multiple round or oval areas of increased density, ranging in size from that of a millet seed to that of a cherry. They were more pronounced in the region of the hila.

*Called "chronic progressive pneumonia" in this country.

These findings were suggestive of metastatic carcinoma, but no primary tumor was discovered. During December the patient had a downward course. He started to complain of pain in the back and legs; x-ray examination of these parts revealed no evidence of tumor invasion. The patient suffered from frequent cough, felt increasingly weak, and had, at times, slight fever. He was referred to a convalescent home on Jan. 10, 1939. A few days later he went home. Unfortunately, exact data could not be obtained as to the last weeks of his illness. His wife stated that the symptoms mentioned above gradually grew worse and that he was running fever and complaining of pain in different regions of the body. He died on Feb. 7, 1939. The clinical diagnosis at the time of his discharge from the hospital was carcinomatosis of the lungs of undetermined origin.

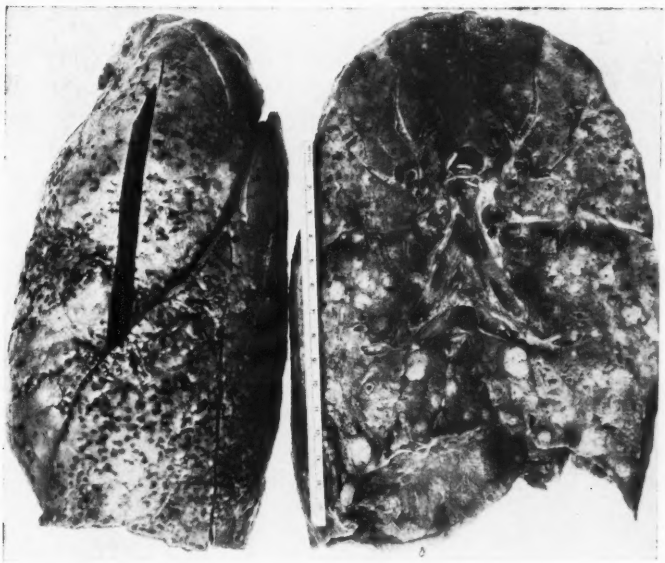

Fig. 1.—Alveolar cell tumor, showing multiple discrete foci.

We did not learn of the death of the patient until after burial. Permission for an exhumation was obtained. Due to excellent embalming and prevailing cold weather the body was amazingly well preserved. The main lesions were found to be present in the lungs. The weights of the left and the right lungs were 970 and 1,030 Gm., respectively. There were but a very few adhesions on the right lower lobe. The lungs were very voluminous and of firm consistency. In many places yellowish tumor nodules were bulging on the surfaces of the pleura, especially on the lower lobes. The pleura exhibited a few scarlike indentations. On section the bronchi and vessels were found to be intact. Only a few aerated regions were present, especially on the base of either lung. Many tumor nodules were noted, measuring 0.2 to 1.0 cm. in diameter (Fig. 1). They were chiefly roundish, rather firm, and yellowish white to yellowish red in color. Part of them exhibited sharp borders; others were confluent. The surrounding lung tissue was chiefly brownish gray or reddish, very firm, somewhat dull, slightly granulated, obviously due to pneumonic lesions; in places it was softer, light gray, and moist. Tumor metastases were revealed in the mediastinal and periaortic lymph nodes, the liver, and the kidneys. Except for the lungs there was no evidence of a primary tumor in

any organ. The gastrointestinal tract, especially, was found to be normal. (Microscopically there was a moderate degree of chronic gastritis, with slight atrophy of the mucosa.) Otherwise the autopsy findings were not remarkable.

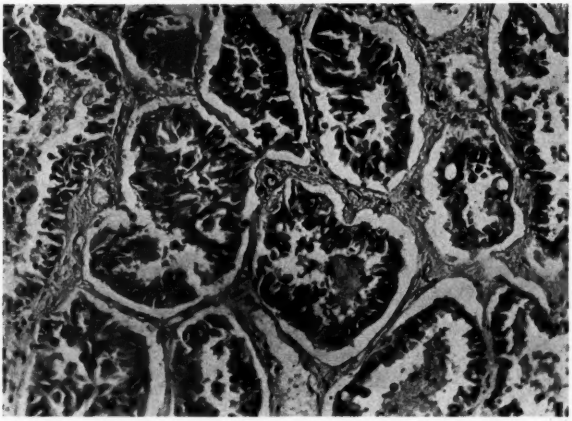

Fig. 2.—Alveolar cell tumor, showing intra-alveolar growth.

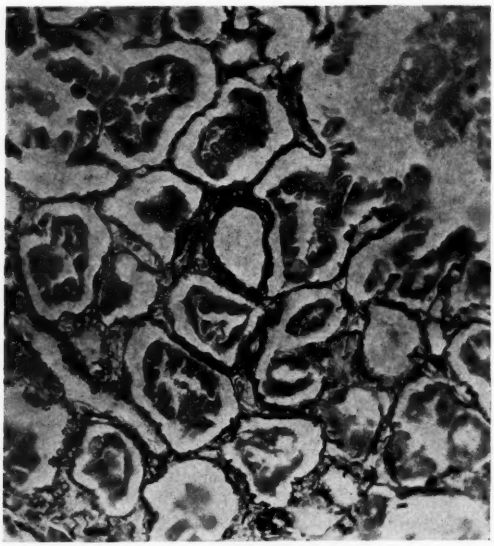

Fig. 3.—Alveolar cell tumor, showing well-preserved elastic fibers in the alveolar walls.

Microscopic examination of the tumor showed that in most foci the alveolar frame was well preserved. The alveoli were lined by one or more layers of cuboidal, partly cylindrical, nonciliated tumor cells (Fig. 2). The cells were adherent to the alveolar walls or were desquamated into the lumina, frequently in the form of continuous rows. (This condition may have been due to autolysis.)

Sometimes a papillary growth into the alveolar lumen was noted. The nuclei were elongated or oval but often irregular in shape and markedly variable in size. There were occasional giant nuclei with bizarre forms. Frequent mitosis was noted. The alveoli containing tumor tissue were quite different in width. The connective tissue of the alveolar walls was partly thickened; the elastic fibers generally proved to be well preserved (Fig. 3); in places there was an increased amount of such fibers. Reticulum fibrils were not present in the tumor tissue. Fibroblasts, mononuclear cells, lymphocytes, and plasma cells were found occasionally in the alveolar walls. Spreading of tumor tissue by way of the pores of Kohn was extremely rare. In some of the foci the alveolar structures were no longer clearly marked, and the tumor exhibited a more irregular growth, resembling an ordinary adenocarcinoma. The lumina sometimes contained mucoid and necrotic masses. Very seldom a fibrous organization of such masses was observed. In the lumen of many alveoli one would find both alveolar phagocytes and desquamated tumor cells. The walls under such conditions would appear devoid of lining cells. For the most part, it was easy to distinguish the phagocytes from the lining cells. The former exhibited a pinkish, vacuolated or granulated cytoplasm and small, dark-stained nuclei, whereas the tumor cells had a more reddish and homogeneous cytoplasm and large, irregular nuclei with several particles of chromatin. Transitional stages were not infrequently seen, and it was difficult at times to discriminate between a phagocyte and a tumor cell. In a very few instances dust and coal pigments were discovered in cells which were indistinguishable from tumor cells. In the tissue surrounding the foci a few emphysematous areas were noted. Elsewhere there was carnification following pneumonia. Many alveoli contained ordinary phagocytes and failed to show lining cells. Sometimes a single alveolus within quite normal tissue was found to be lined or partially lined with tumor cells. In some places fibrous strands grew into the tumors, compressing and replacing the tissue and outlining irregular tumor tubules with flattened low epithelium and inconspicuous nuclei. Such fibrous areas were mainly in the vicinity of the pleura, leading even to grossly visible indentations. Calcification of necrotic tumor cells was rarely observed in such areas. The vessels appeared normal; the vascularity of the tumor tissue was slight. There was no evidence of primary bronchial or bronchiolar involvement, and no epithelial ingrowth from the bronchioles into the alveoli was noted. The bronchi, however, exhibited chronic inflammatory changes, thickened epithelium, and traces of metaplasia into squamous cell epithelium. A few branches showed bronchiolitis obliterans. Very rarely was there circumscribed erosion of bronchial walls by tumor cells located on the outer surface. The metastases showed features similar to those observed in the lung tumors. The origin of the malignant growth appeared to be multiple and primary in the alveolar walls.

DISCUSSION

The author has seen three similar cases in Europe, but complete data are not available for publication. All these cases, including the one reported herein, resembled grossly and microscopically those of Weissmann, Reuss, Oberndorfer, Ewing, Casilli and White, and others; and histologically, also those of Sweany and Bonne.

If the origin of such growths in the alveolar walls is to be proved, the normal cellular conditions in these walls and possible precancerous reactions must be discussed. The so-called alveolar epithelium could be considered to be the starting point of malignant growths. However, the presence of such epithelium in the normal adult life is questioned.

The literature concerning this phase is immense. We restrict ourselves to the quotation of some recent papers. Miller, in his well-known monograph on the lungs, said that the evidence points to a continuous epithelium lining the pulmonary alveoli; he concedes, however, that under normal conditions it is hardly recognizable. Macklin, in summarizing the results of a round table conference on pulmonary alveolar epithelium, stressed that the older idea of the "non-nucleated plate is disappearing and that the number of those denying the presence of alveolar epithelium is increasing." Barnard and Day stated that the epithelial lining of the alveoli "begins to disappear toward the end of the 5th month of embryonic life and does not reappear." The writers stressed that the lung is primarily not a glandular but a vascular organ. Loosli explained that the alveolar lining in the lung of adult mammalians is composed of isolated cells, "septal cells."* They are located in some of the intercapillary spaces; the alveolar membrane which covers the capillaries is of connective tissue origin.

According to our own experience, a cuboidal alveolar lining is present only in early fetal life but disappears toward the end of it and is never seen in the normal adult lung. Such a lining, however, is frequently observed under diseased and experimental conditions, a fact well known to pathologists, repeatedly discussed in the literature, and mentioned in textbooks such as MacCallum's. Lining cells may be noted, for instance, in pneumonia, particularly in "alveolar cell pneumonia" (in psittacosis; Guethert), in lipoid pneumonia (Graef, Freiman, Engelberg, and Merritt), and in collapsed and carnified areas. Our pathologic pulmonary material shows that lining cells are frequently present. In rare cases they may be found in such quantity and arrangement as to suggest early tumorous growth. Oberndorfer[b] described a case of septic-hemorrhagic pneumonia in which many small, yellowish foci were found in the lungs. Histologically the alveoli were lined with high cuboidal epithelium, sometimes producing papillae. Bronchioles were normal. The author assumed a mutation, a sudden rise of a new species of cells displacing the original epithelium. He believed that these changes formed during the course of the pneumonic process, since inflammatory exudate within the involved alveoli was identical with that in other alveoli, and that the metaplasia was a precancerous stage. In so-called chronic progressive pneumonia, called jagziekte in South Africa, a characteristic intra-alveolar proliferation, strongly resembling an adenomatous growth, was described (Cowdry and Marsh, Dungal, and Bonne). We have had the opportunity to compare slides of Oberndorfer's case with those of chronic progressive pneumonia in sheep and were impressed by the striking similarity be-

*This designation was introduced by Lang, in 1925; most authors believe these cells to be mesenchymal in origin.

tween these conditions. Again, both conditions appear to be forerunners of the tumor described in our case, which is in agreement with Bonne's views as quoted above.

If lining cells are absent in the normal adult life, how, then, are we to explain their origin and nature in all these conditions? We begin with the supposition that the normal alveolar walls, according to Maximow and Bloom, Lang, Loosli, and others, consist of a capillary network with perivascular "septal cells," probably of mesenchymal origin and contained in a connective tissue ground membrane. In the first stages of the production of lining cells the septal cells become more numerous and enlarged, and the nuclei become more prominent. Gradually they assume a low cuboidal appearance. There is usually, but not always, a thickening of the alveolar walls; there may be hyperemia and an increased amount of connective tissue with fibroblasts and occasional hematogenous cells. The cuboidal cells within the walls gradually form a continuous lining which becomes definitely demarcated from the other tissue. Desquamation of these cells may eventually occur. The whole process recalls very definitely what Grady and Stewart recently described, in a very thorough and convincing way, with regard to the development of experimental pulmonary cancers in mice. Phagocytes are not infrequently found in close relationship with the lining cells—a fact which points to a common origin. Some microscopic findings as described above suggest the possibility that lining cells may be converted into phagocytes. On the other hand, we found no evidence that phagocytes originated from a pre-existing alveolar epithelium.

As to the further outcome of lining cells, several possibilities may be taken into consideration. (1) In acute or subacute processes, they may be desquamated and disappear with resolution. (2) In more chronic processes (chronic progressive pneumonia in sheep), the hyperplastic areas may grow and eventually form large, multiple adenomas which even may be cystic-papillary in character. (3) The lining cells occasionally found in deformed persistent alveoli, in sclerosed areas, may remain unchanged for an unlimited time. (4) The lining cells may give rise to a malignant growth, the alveolar cell tumor, as described in this paper. This tumor, in its early developmental states, strongly resembles the new growth in Oberndorfer's case, the "adenomas" in chronic progressive pneumonia, the experimental cancers in mice. The morphologic features point to a close relationship between all these conditions (see Bonne also). Thus it appears that a continuous developmental line exists between an occasional production of lining cells in small areas, on one hand, and the alveolar cell tumor, on the other; and one might conclude that lining cells found in all these conditions are of the same origin, i.e., probably mesenchymal.

However, at this point arises the question, can mesenchymal cells produce tumors that morphologically resemble adenocarcinoma? Obviously the attempt to classify the conditions under discussion meets with great difficulties and illustrates the uncertainties in drawing histogenetic conclusions on the grounds of histologic findings. The writer does not endeavor to give a definite solution of this problem; he restricts himself to state that the real nature of those conditions is still open to question. Neither their epithelial nor their mesenchymal character can be proved. Considering the modern views on the development of the lung, the histologic features in the formation of lining cells and their independence of epithelial ingrowth, one finds it difficult to assume that they are of epithelial character. On the other hand it is hardly conceivable that those cellular structures are mesenchymal, since reticulum fibrils were not found, and since the histologic appearance is consistent with that of ordinary adenomas and cancers, respectively. Because of this uncertainty, the indifferent and unprejudicing name of "alveolar cell tumor" was provisionally applied for this neoplasm. The difficulty of the problem is illustrated by the statement of Arkin and Wagner: "There can be no carcinoma primary in the alveoli, as they have no epithelial lining."

The special etiology of the alveolar cell tumor is by no means established. Pneumonia or influenza and the like may elicit in certain people the primary formation of the alveolar lining, but it is unknown why this eventually would degenerate to malignancy. Bonne's theory of a virus causing both jagziekte in sheep and "carcinosis" in man is interesting and suggestive but still lacks definite proof. The readiness with which alveolar cell tumor is produced by poisoning in mice (Grady and Stewart; see here older literature) should be stressed, but it is open to question how far such experimental results are applicable to human conditions.

The clinical diagnosis of alveolar cell tumor may be possible on the grounds of the characteristic x-ray picture: multiple intrapulmonic tumor nodules, no primary tumor elsewhere; chief incidence in middle-aged males.

SUMMARY

1. A case of primary multiple "alveolar cell tumor" in the lung is described.

2. An attempt is made to trace the origin of the tumor cells from the septal cells of the alveolar walls.

3. The relation between the tumor and other conditions showing formation of alveolar lining, in both man and animals, is stressed.

The writer wishes to express his indebtedness to Dr. S. Oberndorfer, Professor of Pathology in Istanbul, Turkey, and to Dr. C. L. Davis, Branch Pathological Laboratory, Bureau of Animal Industry, U. S. Department of Agriculture, Denver, Colorado, for microscopic slides.

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THE SURGICAL TREATMENT OF RECURRENT IDIOPATHIC SPONTANEOUS PNEUMOTHORAX

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ACCORDING to Kjaergaard, who has made the most extensive modern study of the subject, the clinical entity "spontaneous pneumothorax in the apparently healthy person" covers every case of spontaneous pneumothorax that appears without any demonstrable cause in healthy persons in whom tuberculosis cannot be demonstrated either by examination of the sputum, by ascultation, or by x-ray examination, and in whom the lesion is not accompanied by fever or by pleural effusion. It is the opinion of this investigator that the disease is most frequently due to rupture of a subpleural valve vesicle, two types of which he describes. In one, the scar tissue vesicle develops most frequently in the apices adjacent to healed tuberculous areas; in the other, the emphysematous valve vesicle occurs on the basis of local emphysematous changes without scar tissue. In either type, the rupture is due to air entering a subpleural space more rapidly than it leaves so that there is gradually built up a pressure sufficient to rupture the vesicle wall.[2, 3] Rarely, congenital cysts,[4] chronic emphysema,[5] and rupture of pleural adhesions[6] may produce a spontaneous pneumothorax. Occasionally, no abnormality of the lungs can be demonstrated, and the source has remained unknown even in some patients who have died of spontaneous pneumothorax.[7-9]

Nontuberculous spontaneous pneumothorax is an uncommon, but by no means rare, disease and may occur at any age or in either sex, but it most frequently appears in young male adults.[10]

In general, the diagnosis is dependent upon the use of roentgenography, because the condition symptomatically may resemble very closely other intrathoracic or intra-abdominal conditions, and the mild cases may be without symptoms, the condition being revealed only by routine x-rays of the chest.

The course is usually benign, afebrile, and without exudate. There is a definite tendency to spontaneous recovery, the duration of the disease being roughly proportional to the extent of the pneumothorax.

Several possibilities of complications are present, the appearance of which may alter the benign course of the disease. The most frequent of these is recurrence which takes place in from 20 to 45 per cent of

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cases[11] and may appear at any time during convalescence before complete absorption of the air from the previous attack or after complete absorption.[1, 10] The same or the opposite side may be the source, so that bilateral and alternating types are seen, and recurrences have been reported as often as eighteen times in one patient.[12]

Tension pneumothorax with mediastinal shift is reported in about 10 per cent of the cases.[1] This constitutes the most serious complication and is brought about by the action of a valve mechanism allowing air to enter but not leave the pleural cavity until a positive pressure has been attained. If the proper conditions are present, air may escape from the pleural cavity and distend the mediastinal and subcutaneous tissues.

Occasionally, intrapleural hemorrhage accompanies spontaneous pneumothorax. The blood loss may reach alarming proportions, but fatalities are reported less frequently in the literature than one would expect.

The moderate uncomplicated case requires rest in bed for two to three weeks, followed by a period of light physical activity for three to four weeks and abandonment of strenuous exertion for varying periods up to a year.[1, 10]

In severe cases with tension pneumothorax, emergency treatment is required. It is usually sufficient in these patients to perform repeated aspirations of air, although, if the bronchopleural fistula is sufficiently large, a closed intercostal thoracostomy may be necessary.[13]

Attempts to prevent disabling and dangerous recurrences of the condition have led certain persons to inject various irritants into the pleural cavity, so that, when the pneumothorax has been absorbed, the pleural layers will become adherent. The substance most frequently employed is hypertonic glucose,[14-16] although other materials have been utilized,[16] among them the patient's blood.[17] The individual merits of these substances will not be discussed here, although it might be said that the more irritating substances are quite painful for several days, and it seems likely that the less irritating ones would hardly cause sufficient pleuritis to produce adhesions. It is also possible that the effect of even the more irritating substances might have worn off by the time the air had absorbed and the pleural surfaces become apposed.

Appearing in the literature in increasing numbers during recent years are reports of roentgenographic visualization of bullae. Kjaergaard described one case in his monograph.[1] Gordon[18] reported five cases, and Sycamore,[19] one case; recently Castex and Mazzei[20] have added two more. Thoracoscopic evidence of bullae has also been mentioned.[19, 20]

The fact that some of these bullae can be visualized and localized by radiographic and thoracoscopic methods brings up the possibility of a direct surgical approach to the cause of the spontaneous pneumothorax, with resection of the diseased area. In favor of this method is the more

certain cure of the condition, since the lesion itself can be eradicated. Also, pleural adhesions can be more certainly obtained by rubbing the pleural surfaces with gauze and re-expanding the lung at the close of the operation. Conservative measures are uncertain and time-consuming; and, while operative removal is, of course, a major procedure, it seems justifiable in those patients who have been subject to recurrences, and particularly in those where tension pneumothorax is a menace.

Bigger[13] has described one case in which he resected an offending bleb discovered upon exploration of the chest for the site of a bronchopleural fistula. In another instance, Churchill[21] explored the pleural cavity in search of a valve vesicle, but was unable to locate the site of the fistula. He roughened the pleura with gauze, and there has been no recurrence since. We have not, however, found any record in the literature of a bleb having been visualized by roentgenography and subsequently approached directly by surgery.

Dr. L. K. Sycamore,[19] radiologist at the Hitchcock Clinic, previously reported the radiographic appearance of a bleb, substantiated by thoracoscopy and later resected. The surgical aspects were not discussed at that time.

CASE HISTORY

R. S., a white male meatcutter, aged 30 years, was first seen March 30, 1936. Two years previously he had experienced a sudden onset of pain in both sides of the chest, and dyspnea. No roentgenograms were taken. The patient gradually improved and was able to return to work in about two months. A burning, cramping sensation in the right side of the chest persisted for some time. About one year later, he again had a sudden attack of dyspnea and pain, similar to the first. A roentgenogram of the chest at another hospital revealed a complete right pneumothorax, with an emphysematous bleb projecting beyond the border of the collapsed lung. The symptoms improved rapidly, and the patient returned to work in a week. Three weeks later, he had a third attack, and for some time subsequent to this, his physician withdrew air at intervals from his right chest. The third attack lasted four weeks. His symptoms gradually disappeared, and he remained well until the middle of March, 1936, when he suffered a fourth attack of right-sided chest pain and severe dyspnea. He was in bed for two weeks, with only partial relief of the symptoms. At this time, he was referred to the Hitchcock Clinic, and from there was admitted to the Mary Hitchcock Memorial Hospital on March 30, 1936.

On admission, the patient presented a picture of extreme dyspnea and cyanosis, with shallow, rapid respiration and weak, thready pulse. The temperature was normal. Physical examination revealed the signs of a right pneumothorax; roentgenograms showed a right tension pneumothorax (Fig. 1). The patient's acute symptoms were relieved by aspiration of air from the right pleural cavity, but repeated aspirations were necessary in order to keep the dyspnea under control. Because it was felt that the adhesion shown in Fig. 1 might play some part in preventing closure of the perforation in the lung, thoracoscopy was performed on April 11, 1936, and a cautery pneumonolysis was done, severing the adhesions. Subsequent to this procedure, the patient's condition showed progressive improvement, and the lung partially re-expanded. It was at this examination that the large bleb on the right upper lobe was first noted by us (Fig. 2), although its outlines are prob-

ably discernible in Fig. 1. The patient was so much improved symptomatically that he refused to remain in the hospital and was discharged against advice on April 24, 1936.

He returned to work and felt well until May 15, 1936, when he had a fifth attack characterized by dyspnea and cyanosis. He was readmitted to the hospital on May 16, 1936, in essentially the same condition as on the first admission, except that the symptoms were somewhat less severe. The roentgenogram taken at this time showed a right pneumothorax. Another adhesion had formed.

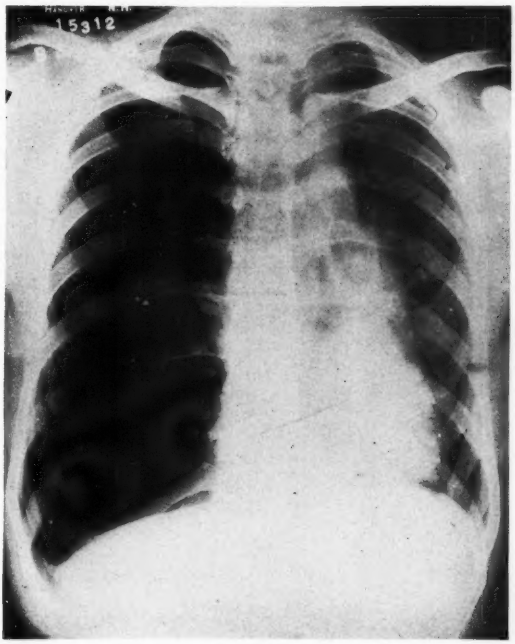

Fig. 1.—X-ray appearance of the chest on admission, showing right spontaneous pneumothorax with mediastinal shift to the left.

The emphysematous bleb was considered to be the cause of the pneumothorax, and excision of this lesion was proposed. Justification for this procedure was based on the fact that the frequent recurrences were handicapping to the patient, and also at least one of the attacks had produced a tension pneumothorax of such alarming proportions that his life was in danger.

Operation was performed on May 23, 1936, under intratracheal gas oxygen ether anesthesia. A right parascapular incision was made, and the fifth rib was resected. The pleura was incised in the rib bed. A bleb, measuring approximately 5 cm. in diameter, was seen on the lateral aspect of the apex of the upper lobe. A broad adhesion extended from the medial aspect and apex of the cyst to the mediastinum. There was a charred area on this adhesion from previous cauterization. There were some adhesions between the middle, upper, and lower lobes, and some between the lower lobe and the chest wall. The pleural surfaces were otherwise normal in appearance.

The adhesion to the cyst was cut away and hemostasis secured. The apex of the cyst was grasped with forceps, and a purse-string suture was placed in the thickened pleura around the base of the cyst. The suture was drawn tight and the bleb removed. The cut surface was sewed over, and the incision in the chest wall was closed without drainage.

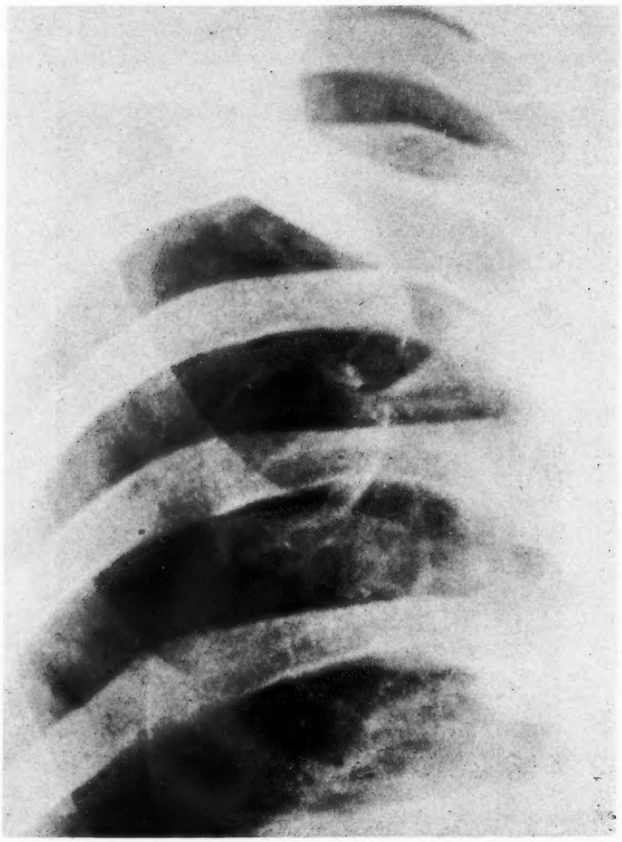

Fig. 2.—Enlargement showing the emphysematous bulla.

The pathologic report was as follows: "Study of numerous sections reveals no definitely indentifiable lung substance. The only feature suggesting the association of this specimen with the lung is the presence of a great many macrophages filled with black, carbonlike pigment. Elastic tissue stains reveal a very small amount of elastic tissue but none in any position to suggest alveolar arrangement. No bronchial structures are seen. Two types of surface are seen in the section; one, which is interpreted as being the original pleural surface, has underlying it a broad band of dense hyalinized fibrous tissue. No covering is apparent, and there is no infiltration of this region. Opposite this surface is one which bears a small amount of inflammatory exudate in spots. Adjacent to this surface there is abundant infiltration by eosinophiles, and a few polymorphonuclear leucocytes and scattered small accumulations of lymphocytes. There are some small arteries which are occluded.

Vascular channels are numerous, and there are some brown, pigment-filled phagocytes suggesting that there has been hemorrhage into the tissues."

Postoperatively the patient ran an oral temperature reaching about 100° F. daily for two weeks. The pulse was about 120 for three days and then leveled off to 100, dropping to normal with the temperature two weeks after operation. Respirations were not elevated.

During this period there was some mucopurulent sputum. Fluid accumulated in the right pleural cavity, and thoracentesis yielded 350 c.c. and 300 c.c., respectively, on the sixth and ninth postoperative days. The fluid was serosanguineous and was sterile on culture.

The patient gradually improved and was discharged on July 1, 1936. He resumed activities and returned to work. He has now been working three years and seven months as a clerk in a grocery store, with no symptoms or ill effects.

SUMMARY

The literature concerning recurrent idiopathic spontaneous pneumothorax is reviewed briefly, and a case is described in which an emphysematous bleb, thought to be the cause of the condition, was visualized by x-ray. The surgical removal of the bleb is described, and the reasons for resorting to radical surgery are discussed.

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COMPARATIVE ACTION OF SULFANILAMIDE, SULFAPYRIDINE, SULFATHIAZOLE, AND ACETYLSALICYLIC ACID (ASPIRIN) ON GROWTH OF THE TUBERCLE BACILLUS IN VITRO

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IN 1938 Ballon and Guernon[1] reported that sulfanilamide (para-amino-benzene sulfonamide) added in certain concentrations to the nutrient media exerted an inhibitory effect upon the growth of the tubercle bacillus. The medium used in their observations was an egg-glycerin-peptone-starch-agar-dye composition recommended by Guernon[2] for the rapid cultivation of the tubercle bacillus. The inhibitory effect noted upon the growth of the tubercle bacillus was in direct proportion to the concentration of the sulfanilamide in the nutrient media. The hydrogen-ion concentration of the media was adjusted after addition of the drug.

The sensitivity of the tubercle bacillus to slight changes in the hydrogen-ion concentration of its environment is well known. It is possible, therefore, that it was this effect rather than any specific quality of the sulfanilamide which may have accounted for the findings referred to above. Other drugs of the benzene ring series might be expected to have a similar inhibitory effect. The inhibitory effects of acetylsalicylic acid (aspirin) and of sulfapyridine and sulfathiazole have been determined in this study, and the observations of Ballon and Guernon[1] with sulfanilamide have been confirmed.

METHOD

The nutrient medium used in the study was that described by Guernon[2] in 1934. The drugs were added to the warm peptone-starch-agar solution in dilutions of 1:100, 1:1,000, 1:10,000, and 1:100,000. Before the addition of the glycerin-egg-dye mixture, the solution was adjusted to a hydrogen-ion concentration of 7. The medium was then tubed, slanted, and inspissated for two hours at 85° C. on the first day and for one hour on the second day. It is well known that the various drugs are not altered by this amount of heating.

Ten milligrams of a three-week-old culture of tubercle bacilli were weighed carefully and were ground with three drops of bile in a graduated centrifuge, with saline added drop by drop up to 10 c.c. The suspension was stirred for one hour, until a fine emulsion was obtained. It was then centrifuged at low speed for three minutes to remove a few large particles. A hanging drop showed uniform distribution of the bacilli. Two drops of this suspension were seeded upon each tube of the medium. Ten tubes were used for each dilution of each drug studied, making forty tubes for each of the four drugs. Forty tubes were used as controls, the medium having

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been prepared without the addition of the drugs. All the tubes were incubated at 37° C. and were sealed with rubber stoppers.

TABLE I

INHIBITORY EFFECT OF VARIOUS CONCENTRATIONS OF SULFANILAMIDE, SULFAPYRIDINE (SODIUM SALT), ACETYLSALICYLIC ACID (ASPIRIN), AND SULFATHIAZOLE IN VITRO ON GROWTH OF THE TUBERCLE BACILLUS

DRUG	DILUTION	NUMBER OF TUBES POSITIVE AFTER INOCULATION OF MEDIA								
Sulfanilamide	1:100	0	0	0	0	0	0	0	0	0
	1:1,000	0	0	0	5	5	5	5	5	10
	1:10,000	0	0	10	10	10	10	10	10	10
	1:100,000	0	0	10	10	10	10	10	10	10
Sulfapyridine (sodium salt)	1:100	0	0	0	0	0	0	0	0	0
	1:1,000	0	0	0	0	0	0	0	6	10
	1:10,000	0	0	10	10	10	10	10	10	10
	1:100,000	4	10	10	10	10	10	10	10	10
Acetylsalicylic acid (aspirin)	1:100	0	0	0	0	0	0	0	0	0
	1:1,000	0	0	0	0	0	0	0	0	0
	1:10,000	0	0	0	0	3	8	10	10	10
	1:100,000	1	3	3	10	10	10	10	10	10
Sulfathiazole	1:100	0	0	0	0	0	0	0	0	0
	1:1,000	0	0	0	0	0	0	0	0	0
	1:10,000	0	0	0	0	0	0	0	0	0
	1:100,000	0	0	0	0	10	10	10	10	10
Controls (40 tubes)		38	40	40	40	40	40	40	40	40
Incubation period in days		13	14	15	21	25	26	28	63	100

TABLE II

DIRECT BACTERICIDAL AND BACTERIOSTATIC EFFECTS OF DRUGS ON SALINE SUSPENSION OF TUBERCLE BACILLI, FIVE TUBES EACH. ALL CONTROL TUBES POSITIVE IN TWENTY-THREE DAYS

DRUG	DILUTION	NUMBER OF TUBES POSITIVE FOUR WEEKS AFTER INOCULATION OF MEDIA									
		TIME OF EXPOSURE IN HOURS OF SALINE SUSPENSION OF TUBERCLE BACILLI TO DRUG DILUTIONS									
		1/6	1/3	1/2	1	2	4	5 1/2	25	48	72
Sulfanilamide	1:100	5	5	5	5	5	5	5	5	5	4
	1:1,000	4	5	5	5	5	4	5	5	5	5
	1:10,000	5	5	5	5	5	5	5	5	5	5
	1:100,000	5	5	5	5	5	5	5	5	5	5
Sulfapyridine	1:100	2	5	4	1	1	3	4	5	5	0
	1:1,000	0	0	5	4	5	5	5	5	3	5
	1:10,000	1	0	5	0	5	5	5	5	4	5
	1:100,000	2	0	3	1	1	5	5	5	3	4
Acetylsalicylic acid (aspirin)	1:100	0	0	0	0	0	0	0	0	0	0
	1:1,000	0	2	0	0	0	0	0	0	0	0
	1:10,000	2	3	5	0	0	0	0	0	0	0
	1:100,000	1	5	5	5	5	5	2	0	0	0

Bactericidal effects of the drugs in various dilutions were also observed by direct contact of the organisms with the drug. A suspension of tubercle bacilli was prepared in the same manner as for the above observations, and the drugs in solution were used in the same dilutions. To 5 c.c. of drug dilution, 0.5 c.c. of the saline suspension of tubercle bacilli was added. At definite time intervals, varying from

ten minutes to seventy-two hours, 4-mm. loopfuls of the suspension exposed to the drug dilutions were transferred to tubes of the drug-free medium; the tubes were incubated at 37° C. and observed daily. Controls, two tubes each, were set up as follows: 5 c.c. of drug dilution were added to 0.5 c.c. of sterile saline, and a loopful of this was seeded on tubes of drug-free medium. Five cubic centimeters of sterile water were added to 0.5 c.c. of the suspension of tubercle bacilli. A loopful of this mixture was transferred to each of the control tubes of drug-free medium. All the controls were incubated at 37° C. and observed daily.

RESULTS

Table I demonstrates the comparative inhibitory action of the four drugs on the growth of the tubercle bacillus when the drugs were added to the nutrient medium. The inhibitory effect varies directly with the strength of the drug. Sulfathiazole seemed to exert the greatest inhibitory influence, under the conditions of the experiment, followed in turn by acetylsalicylic acid (aspirin), sulfapyridine, and sulfanilamide. All the control tubes, forty in all, showed plentiful growth after fourteen days.

No uniformity of results was obtained in the studies of the direct bacteriostatic or bactericidal effects of the drug dilutions on suspensions of the tubercle bacilli. Clumping of the organisms invariably took place when exposed to the drugs in dilution, and a loopful representative of the tube under observation could not be obtained for seeding on the nutrient medium. In some instances the controls did not grow out, probably because of clumping in sterile water. Taken as a whole, it can only be stated that some inhibitory effect was observed in the case of all the drugs studied, roughly proportional to the time of exposure of the organisms to them, but less in effect and in uniformity than observed after adding the drugs directly to the nutrient medium.

DISCUSSION

The results obtained verify the findings of Ballon and Guernon[1] with sulfanilamide, and demonstrate a similar effect on the part of sulfathiazole and sulfapyridine (sodium salt), as well as acetylsalicylic acid (aspirin). The hydrogen-ion concentration was carefully adjusted in all the experiments, at the time of inoculation of the medium. Possible changes in hydrogen-ion concentration during the period of observation were not studied, as solid media do not readily lend themselves to such determinations.

The mechanism by which the drugs inhibit growth of the tubercle bacilli is obscure, as it is in the case of in vivo experiments reported by Rich and Follis,[3] Ballon and Guernon,[2] Smithburn,[6] Greey and associates,[7] and others. Corper and co-workers[4] believed the effect of sulfanilamide on the spread of tuberculosis in guinea pigs more apparent than real, and thought it best explained as an "organic toxic effect" of the drug.

SUMMARY

Inhibitory action of sulfanilamide, sulfapyridine, sulfathiazole, and acetylsalicylic acid (aspirin) on the growth of the tubercle bacillus in vitro has been noted. The action varied directly with the strength of the drugs, and seemed greater with acetylsalicylic acid and sulfathiazole

than with sulfanilamide and sulfapyridine. The inhibitory effect of these drugs on in vitro growth of tubercle bacilli does not seem to be specific. Other bactericidal and bacteriostatic tests were inconclusive.

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AN HISTORICAL CONSIDERATION OF THE DIAPHRAGM

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THE partition, partly muscular, partly tendinous, which divides the thoracic from the abdominal cavity was referred to by Hippocrates (460-375 B.C.) as "phrenes." Aristotle (384-322 B.C.) called it "hypozoma" and "zoma." To many it was known as "septum transversum" or "disseptum"; to others it was known as "cinetus," and to still others, as "discrimen thoracis et ventris" or "succingens membrana."* It was of interest, therefore, to consider who first employed the term diaphragm for the "Skynne that depárteth and is sette bitwene the bowels and the spirytuall membres" (Bartholomaeus Anglicus, translated by Trevisa in 1397). Galen (A.D. 130-200?) states† that it was through Plato that the term diaphragma came into use, in place of phrenes. It is interesting to note, however, as Dr. Francis, of the Osler Library at McGill University, has pointed out to me, that this does not mean that Plato himself substituted diaphragm for phrenes. The passage in Plato's "Timaeus," 70 in Bury's translation (Loeb Classics, 1930), reads: "And within the chest—or 'thorax,' as it is called—they fastened the mortal kind of soul. And inasmuch as one part thereof is better, and one worse, they built a division within the cavity of the thorax—as if to fence off two separate chambers, for men and for women—by placing the midriff‡ (phrenes) between them as a screen (diaphragma)."§ Evidently Plato merely compares the phrenes to a diaphragma, the latter being the Greek word for partition or screen.

The membranous or tendinous portion of the diaphragm has been variously referred to as "pars nervosa," "centrum nerveum," "centrum tendinosum," "coostrum," "centre phrénique," and by Winslow as the middle aponeurosis or aponeurotic plane of the diaphragm.

Hippocrates was well aware that the diaphragm might become the seat of disease, but apparently did not attribute any movement to it.

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*Others, using the term "praecordia," implied not only the region above the stomach but the diaphragm as well.

†*De locis affectis*, book 5, chap. 4.

‡The earliest recorded use of the English term midriff is in the Anglo-Saxon "Leechdoms" about A.D. 1000. In the printed edition (vol. 2, 1865, p. 179), the passage is thus translated: "At whiles it beginneth from the midriff, which is betwixt the wamb [belly] and liver." This, it may be noted, places the midriff in an anomalous position.

§It should be noted that Hippocrates and other ancients employed the term "diaphragma" to indicate the division between the mouth and esophagus ("diaphragma per oesophagum"). Rufus of Ephesus called the cartilaginous partition between the nostrils the diaphragm of the nose.

Aristotle believed that as a result of its density, the central or so-called nervous portion of the diaphragm prevented vapors in the stomach from entering the chest. Other ancients, according to Mondeville (1260-1320), believed that the function of the diaphragm was to protect the "spiritual" members of the body. Still others were of the opinion that the diaphragm served to keep the stomach and organs of nutrition warm. It was suggested by Mundinus that the diaphragm played a part in propelling matter from the bowels. There were also those who intimated that the diaphragm aided in the expulsion of the fetus and in the discharge of liquors contained in the excretory ducts of glands.

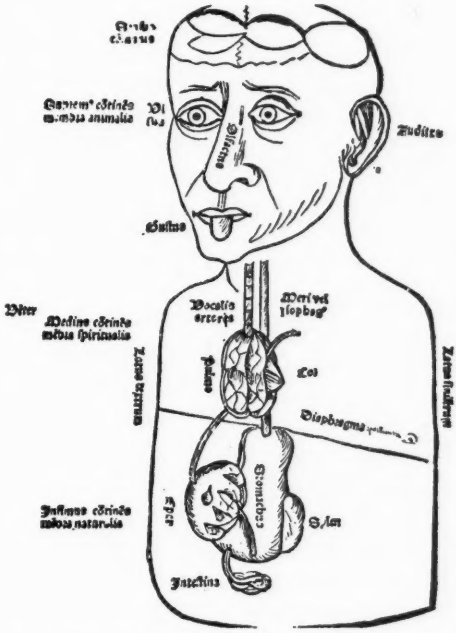


Fig. 1.—Situs figure from Johann Peyligk's "Philosophiae Naturalis Compendium" (Leipzig, 1499). (After Nicaise, Chirurgie de Mondeville, Paris, 1893, p. 49.) This figure is purely schematic, as are those by Peyligk's contemporaries, Ketham (1491) and Hundt (1501). The well-known situs figure from the "Anthropologium, de hominis dignitate" of Magnus Hundt has the diaphragm omitted. In this illustration from Peyligk, the diaphragm is represented as being pierced by the esophagus and the great vein. According to McMurrich, the five-lobed liver with the gall bladder resting on it suggests a derivation from the Five Figure Series. It could also be regarded as a Galenic error.

Before proceeding with physiologic considerations, we might refer briefly to the early anatomic descriptions and figures of the diaphragm. The figure of the muscle system from MS. Ashmole 399 is of interest to us because Sudhoff has questioned whether or not the diaphragm is to be discerned in this figure. This anatomic illustration of the pre-Vesalian period is apparently not based upon actual dissection but upon

earlier traditional diagrams (Sudhoff). The figure by Johann Peyligk (1474-1522), the illustration from Reisch's "Margarita Philosophica" (1503), and the one by Phryesen* (1518) are early examples which not only used the term diaphragma but which placed it in its proper position anatomically. (Figs. 1, 2, and 3.)

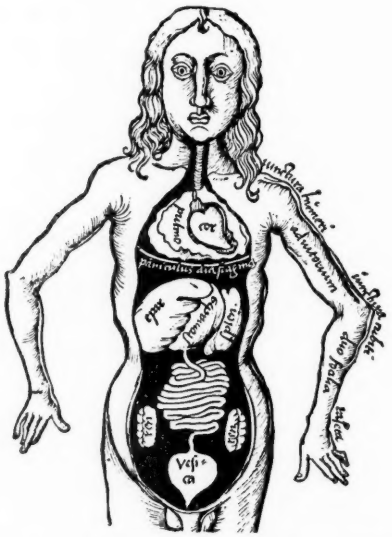


Fig. 2.—Illustration from the encyclopedia "Margarita Philosophica," edited by Gregor Reisch (c. 1467-1525). Note the trachea, the right lung, and heart on the left side. Note also the black background for the thoracic and abdominal organs. The diaphragm is represented as a transverse septum. The esophagus is absent. In other editions similar woodcuts show the words "Corpus phisicum" engraved in capitals at the top, beside the head. (After Johann Ludwig Choulant, English translation by Mortimer Frank, Chicago, 1920, University of Chicago Press.)

As for ancient anatomic descriptions of the nature, origin, and insertion of the diaphragm, Aristotle stated that the diaphragm, with a thin membrane in the middle of it, coming under the lungs, was attached to the ribs, the hypochondrium, and the backbone;† he said further that it had veins running through it, and that the diaphragm in man is thicker in proportion to the size of his frame than is the diaphragm in animals. In this connection it is worthy of note that John Hunter (Works, vol. iv. p. 177) first appreciated that birds have diaphragms. He said: "It has been asserted that birds have no diaphragm; but this opinion must have arisen either from want of observation, or from too confined an idea of a diaphragm; for there is a moder-

*According to Garrison (The Principles of Anatomic Illustration Before Vesalius, New York, 1926, Paul B. Hoeber, p. 43). Phryesen, who is also referred to as Friesen and Frisius, made the first map containing the name "America."

†One obviously incorrect statement by Aristotle, and referred to in the English translation of his works, is that two branches separate off from the vein which passes through the liver, one of which terminates in the diaphragm.

ately strong, but thin and transparent membrane covering the lower surface of the lungs, and adhering to them, that affords insertion to several thin muscles which arise from the inner surfaces of the ribs."

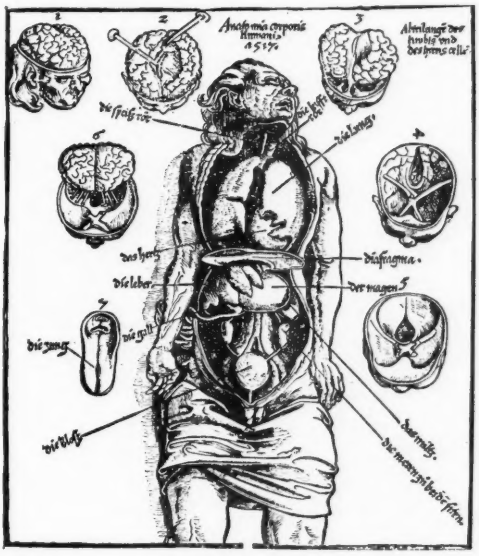


Fig. 3.—Situs figure from the "Spiegel der Artzny" of Laurentius Phryesen (Strassburg, 1518) after Choulant. This figure is said to represent the anatomy which originated with Mundinus in the Middle Ages. In this figure there is evidence of greater accuracy in the representations of the viscera. The thoracic and abdominal cavities have been cut open. The six small diagrams show the anatomy of the brain. There is also a picture of the tongue. Note the five-lobed liver. The heart lies in the median line of the body with its axis to the left; it is surrounded by the left lung. The right lung has only two lobes. The trachea, heart, and lungs have been drawn in relatively correct proportions. The diaphragm appears as a horizontal disklike structure, with the liver under it on the right, and the stomach on the left. The kidneys, which lie very low, are joined by short ureters to the bladder, which lies very high. Above the plate are cut the words: "Anatomia Corporis Humani 1517." Blumenbach attributes the woodcut to a pupil of the elder Holbein.

Galen (A.D. 130-200?) erroneously described the diaphragm as membranous above and below; nevertheless, he spoke of two muscular extensions which were attached to the xiphoid cartilage. He also drew attention to two natural perforations or hiatuses in the diaphragm, one of which permitted passage for the vertebral column, esophagus, and the great artery, and the other of which received the vena cava. Galen has presented us with a most interesting description of those muscles which, by their action, are antagonists of the diaphragm. He was the first to describe the internal intercostal muscles.

Aurelius Cornelius Celsus (53 B.C.-A.D. 7) has furnished us with an excellent definition of the diaphragm, namely, a transverse partition of strong sinewy membrane which separates the belly from the precordia and through which many blood vessels course. Gabriel de Zerbi (1468-

1505) recognized that the fleshy portions of the diaphragm were situated at the circumference or periphery, and the membranous or tendinous portion in its middle.

Andreas Versalius* (1514-1564) "likened the shape of the detached diaphragm to a sting ray." He erroneously stated that Galen had described the esophagus as passing behind the diaphragm. Actually, both noted the opening for the passage of the esophagus through the diaphragm on the left and the opening for the vena cava on the right (Fig. 4).

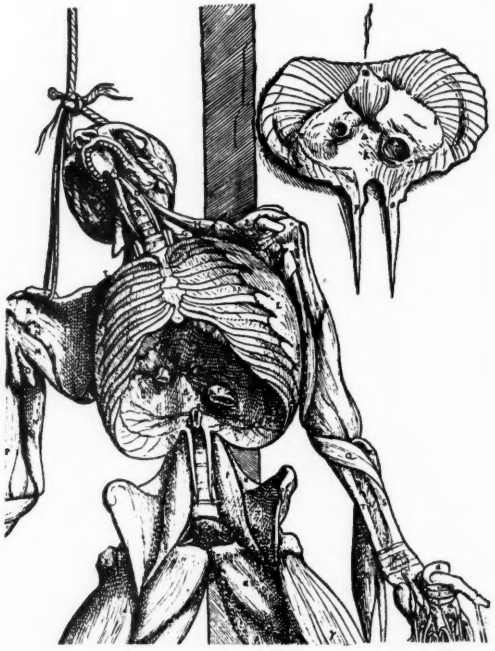


Fig. 4.—From Andreas Vesalius' "De humani corporis fabrica" (Basle, 1543). The illustrations of the first edition of this work are said to have been drawn by Jan Stephan, called van Calkar. A first edition was presented to McGill University Medical Library by Sir William Osler in 1909. In the above Septima Musculorum Tabula, the undersurface of the diaphragm is in view. In the insert, an excellent picture of the diaphragm, the crura, and the musculous and tendinous portions, as well as the apertures for the inferior vena cava and esophagus, are clearly indicated.

It is difficult to refrain from inserting here the fine panegyric on Vesalius' book which Osler inscribed on March 9, 1909, in the copy of the first edition which he presented to McGill University, his old school.

"In itself, for what it contains, but still more for what it did, the 'Humani corporis fabrica' is one of the great books of the world, with which in the literature of Medicine only the 'De motu cordis' of Harvey is to be compared. The one revolu-

*It is worthy of note that in the experimental animal, in other words, in one with the chest open, in the presence of open pneumothorax Vesalius kept it alive by forcing air into the trachea by means of a bellows.

tionized anatomy, the other created modern physiology. This work of Vesalius is the first modern treatise on anatomy based upon dissections of the human body. To appreciate the extraordinary character of the descriptions and of the plates they must be compared with contemporary works. Vesalius really described the body as we know it, for the first time fully, and for the first time accurately. It is difficult to say whether in text or figure the departure from the anatomy of the day is more striking. There are grave mistakes of omission and of commission, but they appear insignificant in a volume full of such important contributions. To the middle of the sixteenth century anatomy was taught from the writings of Galen, not from what was seen in the occasional public dissections. From what the Great Master had written there was no appeal, and the veneration with which his works were regarded was as for a Gospel, like the feeling men have towards the sacred Scriptures. Imagine the surprise and the consternation of the easy-going old professors who held the chairs of anatomy to have a huge volume thrust into their hands filled from cover to cover with descriptions and figures with which they were unfamiliar. And written by a young man of twenty-eight! Headed by his old teacher Sylvius a storm of opposition soon raged, and a vindication of Galen was attempted, but it was soon found that the old anatomy, correct enough in places, was largely that of swine, dogs and monkeys, while the 'Fabrica' contained descriptions and figures from human dissections. To understand the phenomenon, almost unique in the history of science, of a revolution of this character effected by so young a man, we must remember that from boyhood Vesalius had had a perfect passion for dissecting. After devoting his energies to the anatomy of the domestic animals, he robbed graveyards and the gallows for human skeletons, while as early as 1534-36, as prosector to Sylvius and Guinterius in Paris, he had opportunities to dissect the human body. His reputation must have been remarkable, as at the age of twenty-three he was appointed Professor at Padua, one of the leading schools of Europe. The 'Fabrica' remains a monument of human effort, one of the greatest in the history of our profession." (William Osler, Rome, March 9, 1909.)

Nicolas Habicot (1550-1624), in his "Paradoxe Myologiste," referred to by Portal, expressed the belief that the diaphragm is not a solitary muscle. This view was contrary to the opinion of most of the ancients and to those of his time.

Caspar Bartholin, son of Thomas, had his important work upon the diaphragm published in 1676. It was entitled "Diaphragmatis structura nova. Accessit methodus praeparandi viscera per injectiones liquorum." His figures of the diaphragm closely resemble those of Vesalius. Bartholin was among those who maintained that the diaphragm was made up of two muscles, one the superior, the other the inferior. He was convinced that its tendinous portion acted as a barrier to its movement. Bartholin stated that the inferior muscle of the diaphragm had two pillars or crura through which the aorta passed. He advanced the opinion that the diaphragm and the transverse abdominal muscles combined to form a trigastric muscle. The diaphragm is in structure, development, and action still regarded as a true digastric muscle made up of two segments, a spinal or dorsal, and a costal or ventral (Keith). Bartholin also recorded that the diverging fibers of the anterior and

superior muscles of the diaphragm formed a triangular aperture underneath the xiphoid cartilage. This had been noted by others. He indicated that, for the most part, the muscle fibers interlaced in such a way as to form different groupings or bundles which extended in various directions. His numerous observations upon the "motor activity" of muscle fibers are interesting. He was of the opinion that all parts of the body, whether solid or fluid, owed their movement to motor fibers which he stated were always fleshy.

Fig. 5.

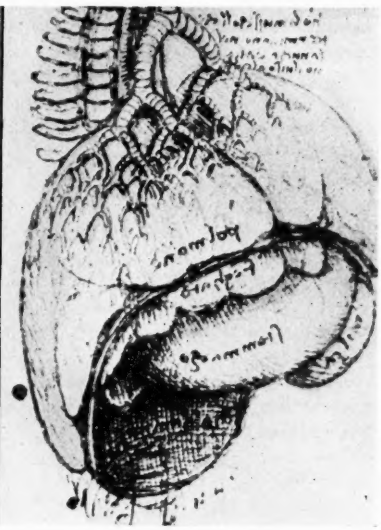


Fig. 6.

Fig. 5.—After Leonardo da Vinci, showing the form of the diaphragm (Q I, 5). Leonardo, according to McMurrich, "believed that if the shoulders were raised by the muscles of the neck, the lowering of the ribs would be prevented and, similarly, if the shoulders were depressed the ribs could not be raised and so, to obviate difficulties, Nature provided the diaphragm to attend to the necessary increase of chest capacity (An B, 16v)." The form of the diaphragm "is that of a deep spoon."

Fig. 6.—Trachea, lungs, diaphragm, liver, stomach, and spleen of an animal (Leonardo da Vinci, An B. 37v.).

If, in the light of our present knowledge, we piece together the descriptions of the origins and insertions of the diaphragm given by Bartholin and those who came before him, it becomes obvious that, save for different terminology and inconsequential details, relatively little was added by the anatomists who came later. That such is the case will be appreciated further by studying the figures illustrative of anatomic dissections of the diaphragm by Leonardo da Vinci (1452-1519) (Figs. 5, 6, and 7), Charles Estienne (1504-1564) (Figs. 8 and 9), and Vesalius (1514-1564) (see Fig. 4).

Galen (A.D. 130-200?) not only appreciated that the diaphragm could undergo changes in shape, but also recognized that its action was con-

Fig. 7.—Sketch of the lungs and heart by Leonardo da Vinci, showing the pleural cavities (Q, IV, 3). Being left-handed, Leonardo developed the art of writing from right to left. This is often referred to as looking-glass or mirror writing.

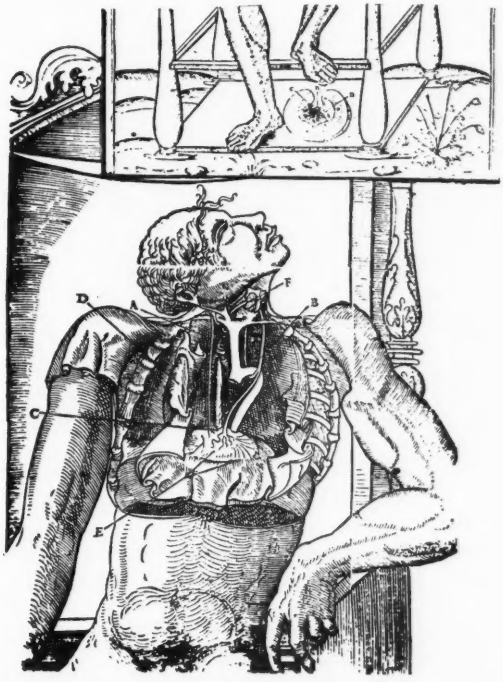


Fig. 8.—The thoracic organs, from Charles Estienne (Stephanus), "De dissectione partium corporis humani," Paris, 1545. In this illustration, the thorax is open and the heart and lungs have been removed. The arch of the aorta is seen with the vagus nerve *A* and *B* on either side, and the recurrent laryngeal nerve on the left. The left vagus nerve below the level of the recurrent laryngeal takes the course of the phrenic nerve and appears to divide over the diaphragm like the phrenic rather than the vagus nerve. The pleura is shown turned back at *D*, and the esophagus at *C* penetrating the diaphragm. In the complete figure, the insert (in the upper right corner) appears below the left foot. This is a poor figure of the diaphragm in which the foramen for the esophagus is indicated by *B*, the aorta by *C*, and vena cava by *D*.

cerned with respiration. He is credited by some with being the first to make this all-important observation. Mundinus (?1276-1326) wrote at length upon the diaphragm; and it is worthy of note that he discussed its action upon the viscera. In this connection, a consideration of the effects of a high, insufficient, or paralyzed diaphragm upon the function of the gastrointestinal tract is of interest, for the diaphragm is the active factor in the production of displacement of viscera. (Figs. 10 and 11.)

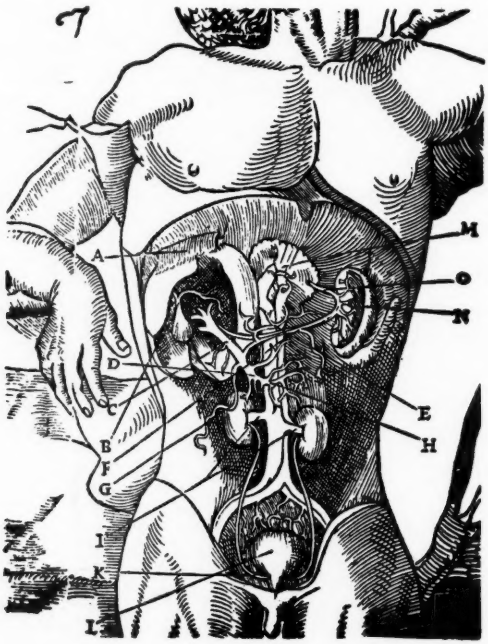


Fig. 9.—This figure shows the undersurface of the diaphragm and some of the contents of the abdominal cavity. *A* represents the ligament between the diaphragm and liver; *B*, the undersurface of the liver and the gall bladder; *C*, the portal vein; *N*, the spleen. *M* denotes the esophagus after it has escaped through the diaphragm.

Hieronymus Fabricius ab Aquapendente (1537-1619) was convinced, and rightly so, that the diaphragm normally maintained a more elevated position on the right side than it did on the left.

Jean Riolan the Younger (1580-1657) regarded the diaphragm from a functional standpoint as the sole muscular agent of respiration, that is, as a muscle which contracted during inspiration and which altered its shape further during relaxation.

Habicot rightly believed that the muscle on one side could become paralyzed, notwithstanding that the other remained healthy. It is worthy of note that it was not until 1911 that artificially induced

paralysis of the diaphragm was recommended as a therapeutic procedure by Stuertz.

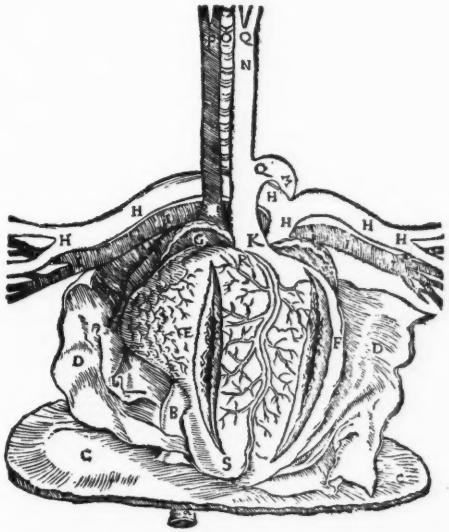


Fig. 10.—From Dryander's "Anatomia Mundini," Marburg, 1541. Mundinus wrote his famous manual of anatomy in 1316. None of the manuscripts are illustrated. Johann Eichmann, called Dryander, is regarded as among the first anatomists who made illustrations after their own dissections. Note the diaphragm *CC*; the involucrum of the heart, a thin membrane, *DBD*, surrounding the heart and filled by its substance; the right ventricle, *E*; left ventricle, *F*; right auricle, *G*; course of the arteria venalis (pulmonary vein), *HHH*; course of the vena arterialis (pulmonary artery), *IIII*; the beginning of the aorta, *KQ*; the aorta ascendens, *QN*; aorta descendens, *QM*; trachea, *O*; great ascending veins, *P*; veins nourishing the heart, *R*; and the extremity of the heart, *S*.

Some original letters of Sir Thomas Browne (1605-1682), collected by Sir William Osler, are in the Osler Library at McGill University. In one of those letters made available to me by Dr. W. W. Francis, who suggested that it was probably written in 1679, is the following interesting note:

Diaphragma

Concerning the motion hereof, I am fayne to make this distich for my memorie:
Contrahit, exque cavo rectum est, dum spiritus intrat,
Erigit, ex Rectoque cavum est, dum spiritus exit.

The anatomist Jean Senac (1693-1770) stated that the central portion of the diaphragm does not descend during inspiration, and also that the lungs in their normal state are everywhere contiguous with the pleura. Senac accurately described the aperture in the diaphragm for the vena cava, noting that under normal conditions, the inferior cava is not compressed by the diaphragm, for the large vein pierces the tendinous portion of the diaphragm. Mittenleiter observed that

the portion of the vein which pierces the diaphragm is practically free from muscle fibers. After it has passed through the diaphragm, it becomes rich in elastic tissue and smooth muscle. Jan Swammerdam (1637-1680) noted that when the diaphragm ascends, it reaches to the level of the fifth and sixth ribs.

Emilio Parisano (1567-1643) suggested that in its motion the diaphragm exerted a light pressure effect upon the aorta, thus helping the flow of blood upward and downward. Others were of the opinion that the movements of the diaphragm depended upon those of the heart.

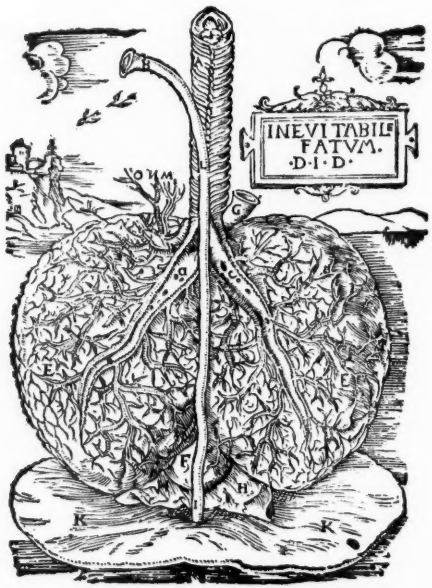
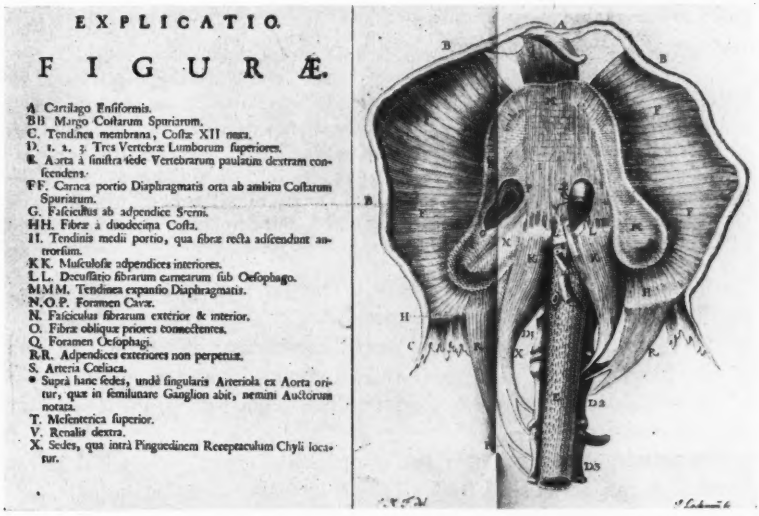


Fig. 11.—The inscription in this figure from Dryander is worthy of note. The lungs appear as spongy organs. The trachea with its cartilaginous rings and the main bronchi opened up are clearly distinguishable, as are the esophagus and diaphragm.

The thoracic support of the diaphragm is afforded by the pericardium, heart, great vessels, and lung which attach the upper surfaces of the diaphragm to the structures at the root of the neck and to the whole extent of the thoracic wall. It should be obvious, therefore, that the diaphragm influences the circulation, just as an enlarged heart may force the diaphragm down posteriorly, a fact noted by Boerhaave (1668-1738).

The action of the diaphragm upon the circulation may be indirect through changes in respiration, or as the result of altered intrathoracic or abdominal pressure, or it may be direct through action on the heart and blood vessels.

Leonardo da Vinci's interest in the spinal nerves was concentrated mainly on those that pass to the limbs. He barely makes mention of the intercostal nerves (An B, 30), "stating that they arise from the spinal cord, the lowest one having its origin at the level of the kidney; and of the nerves from the cervical plexus he figures only the phrenic (An B, 33v and 34), which he traces from the fifth cervical nerve to the pericardium, failing to note its continuation to the diaphragm, although its connection with that muscle had been shown experimentally by Galen and is mentioned by Avicenna" (McMurrich, Leonardo da Vinci, Baltimore, 1930, Williams and Wilkins Co., p. 212).



EXPLICATIO

FIGURÆ.

A Cartilago Enſiformis.
BB Margo Coſtarum Spuriarum.
C. Tendinea membrana, Coſtæ XII nata.
D. 1. 2. 3. Tres Vertebræ Lumborum ſuperiores.
E. Aorta à ſiniſtra ſede Vertebrarum paulatim dextram conſcendens.
FF. Carnea portio Diaphragmatis orta ab ambitu Coſtarum Spuriarum.
G. Faſciculus ab adpendice Sterni.
HH. Fibræ à duodecima Coſta.
II. Tendinis medii portio, qua fibræ recta adſcendunt antrorſum.
KK. Muſculoſæ adpendices interiores.
LL. Decuſſatio fibrarum carnearum ſub Oeſophago.
MMM. Tendinea expanſio Diaphragmatis.
N.O.P. Formen Cavæ.
N. Faſciculus fibrarum exterior & interior.
O. Fibræ obliquæ priores connectentes.
Q. Foramen Oeſophagi.
RR. Adpendices exteriores non perpetuæ.
S. Arteria Cœliaca.
• Suprà hanc ſedes, undè ſingularis Arteriola ex Aorta oritur, quæ in ſemilunare Ganglion abit, nemini Auctorum notata.
T. Meſenterica ſuperior.
V. Renalis dextra.
X. Sedes, qua intrà Pinguedinem Receptaculum Chyli locatur.

Fig. 12.—From Albrecht von Haller, "De Diaphragmatis Musculis Dissertatio Anatomica," Leyden, 1738. Haller's description of the diaphragm is anatomically perfect. He prepared the costal pleura of an animal in such a way as to permit of observations upon the underlying lung. Haller showed that the lung expanded passively and followed the receding thorax and descending diaphragm. He referred to the intercostal muscles as elevators of the ribs.

Giovanni Battista Morgagni of Forli (1682-1771) also drew attention to the fact that the diaphragm was innervated by intercostal nerves. Albrecht von Haller (1708-1777) likewise made interesting observations upon the diaphragm and its nerve supply. (Fig. 12.)

Further remarks upon the innervation of the diaphragm will be concerned only with the observations of Thomas Willis (1621-1675). In his work, "Anatomy of the Brain" (Ch. 28, works, 1681 ed.), one can read the following upon the nerve to the diaphragm:

"As to its beginning it may be observed, that it arises from the brachial nerves with a double or triple root; two or three shoots going out of the aforesaid nerves, grow together in the same trunk, which is the nerve of the Diaphragma. In man its

first shoot, which is also the greatest, is produced out of the second vertebral nerve (cervical); and when the first brachial nerve arises from the same handful of vertebral nerves going out at this place, the aforesaid shoot is rooted in its origin: wherefore, when in Brutes the first brachial nerve arises from the fourth or fifth vertebral, the nerve of the Diaphragma also begins its rise far lower: two other shoots arise out of the same stocks of the brachial nerves which follow next. But the trunk which is made out of these shoots goes forward single through the passage of the neck and the cavity of the thorax without any branching forth, even to the Diaphragma, where being at last stretched out into three or four shoots, it is inserted on either side of the fleshy or musculous part of it."

One will note in the above the author's accurate description of the course of the phrenic nerve within the thorax.

Willis wrote in the following manner concerning the function of the main nerve to the diaphragm:

"Because the Diaphragma is a muscle, and performs both its motions, to wit Systole and Diastole, by its own Fibres, the office of either nerve is only to carry bands or forces of animal spirits requisite for the indiscontinued action of that part, and also to convey thither the instincts of the Motions variously to be performed. As to the first use of this nerve, viz. for the passage of the animal spirits, the business is performed in this muscle as it is in the Heart. The spirits flowing into the Diaphragma by the nerves receive subsidiary Forces, to wit a Sulphureous Copula from the blood, upon whose Explosion being still iterated by turns, and the receiving of new, the action of this perpetual moveable depends."

One may perhaps see in the above a possible hint of the existence of the blood chemical factor in respiration.

The role of the diaphragm in forced respiratory efforts, in the acts of coughing, sneezing, laughing, and in singultus, and a consideration of phrenitis and other diseases of the diaphragm will be dealt with on another occasion.

The classical description of the symptomatology of congenital intrathoracic hernia, published by Fothergill in 1746,* will be the only reference to abnormalities of the diaphragm in this paper. His article appeared in the Philosophical Transactions (No. 478, p. 11, 1746) and was also included by John Coakley Lettsom in his three-volume edition of the "Works of John Fothergill" (1783), together with a translation of this letter on "A Ruptured Diaphragm and a Change in the Situation of Some Viscera Observed in the Body of a Female Ten Months Old," addressed to Richard Mead. We read:

"The respiration of the infant was too frequent from its birth, and a few days after came on a stuffing and heaviness of the head as if from having caught cold, with a considerable excretion of a watery mucus from the eyes, nose and mouth, which as often as it was obliged to suck, almost threatened suffocation. On this account it would cry and go into more violent passions than is usual at so tender an age; so that the attendants would often fear, lest in a short gust of passion it should expire." It was noted that the infant was seized with a sudden vomiting, more frequently than is common to most children. "The matters thrown up neither resembled crude nor digested food, but a kind of purulent pap, sometimes

*Read before the Royal Society, June 16, 1745.

acid, sometimes of a fetid smell; the stools also were too frequent, and exactly of the same kind of matter: the respiration in the meantime frequent, laborious and during the night extremely difficult.''

In conclusion, Fothergill commented thus: ''Everyone skilled in medicine I think will suppose from the history that the disease is a new one. . . . But who would ever have conjectured that the septum transversum was divided asunder?'' The necropsy findings were typical of intrathoracic hernia. The dyspnea, the upper respiratory tract infection which is so prone to occur in such infants and which may extend down into the lower respiratory tract, the embarrassment during feeding, with its ever-present possibility of aspiration pneumonia, the increased dyspnea when the infant assumed certain postures, as well as the signs and symptoms of intestinal obstruction—all of these afford us a most complete clinical picture of an intrathoracic hernia, which in this instance was left-sided.

For the facilities of the Osler Library and for invaluable aid in the preparation of this paper, I am deeply indebted to Dr. W. W. Francis.

HEMOTHORAX: ITS RELATION TO PRIMARY CARCINOMA OF THE LUNG

HENRY LEO CABITT, M.D.
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THERE is a general agreement about the increase in the incidence of primary carcinoma of the lung. The presence of blood in the pleural space has been generally accepted as of diagnostic and prognostic significance in carcinoma of the lung. Nevertheless, careful search of the literature disclosed no assembled group of cases of hemothorax* studied so as to derive basic concepts as to the cause, incidence, and prognosis of this sign. Only isolated case reports of hemothorax noted in the course of various diseases, with the interpretation and progress of this finding, could be found. Our interest was primarily in the relationship of hemothorax to primary carcinoma of the lung, but to clarify such an analysis, hemothorax in general was briefly considered.

CAUSES OF HEMOTHORAX

All cases of primary carcinoma of the lung and of hemothorax and hemohydrothorax in the records of three Boston hospitals were carefully reviewed. A total of 247 cases of primary carcinoma of the lung were accepted as proved. Eighty-six cases with acceptable evidence for hemothorax or hemohydrothorax were also found.

In Table I these eighty-six cases of hemothorax are arranged as to etiology. The commonest cause of this condition is trauma, which accounted for 45 per cent of these eighty-six cases. The trauma consisted of bullet wounds, stab wounds, or crushing injury to the chest. The mechanism of the hemothorax in these instances is obvious. The next most common cause of hemothorax is primary carcinoma of the lung, which accounted for thirty-five (41 per cent) of the cases. These thirty-five cases will be analyzed in detail below. Twelve of the eighty-six patients manifested hemothorax not due to trauma or primary carcinoma of the lung. In three of these the hemothorax was associated with pulmonary tuberculosis proved by sputum or pleural fluid examination. In tuberculosis the hemothorax is due to the breaking of an

From the Thoracic Clinic, Beth Israel Hospital, Boston, Mass.
Read (by title) before the Twenty-second Annual Meeting of the American Association for Thoracic Surgery at Los Angeles, Calif., July 5, 6, and 7, 1939.
*The terms hemothorax and hemohydrothorax will be used interchangeably.

adhesion in which blood vessels are contained or a rupture of the diseased pulmonary parenchyma. There are isolated reports in the literature of similar findings.[1] The treatment and prognosis of hemothorax complicating pulmonary tuberculosis will not be discussed.

TABLE I

ETIOLOGY OF HEMOTHORAX

	NO. OF CASES	PERCENTAGE
Trauma	39	45
Primary carcinoma of lung	35	41
Other causes	12	14
Pulmonary tuberculosis	3	
Cardiac disease	2	
Sarcoma of ribs	2	
Metastatic carcinoma of lungs	2	
Banti's disease	1	
Lymphosarcoma of mediastinum	1	
Lobar pneumonia	1	

One of the two patients with cardiac impairment complicated by hemothorax had cardiac decompensation, mitral valve disease, and possible infarction of the lung; in the other patient, autopsy revealed rheumatic heart disease with mitral stenosis.

Two persons had metastatic carcinomatous involvement of the lungs, pleura, and pulmonary lymph nodes, as proved at autopsy. In one, the primary focus of the neoplasm was the breast; in the other, the pancreas was the original site. Thus, although secondary metastatic carcinoma of the lung occurs more frequently than primary carcinoma of the lung, in this series the combination of such metastasis and hemothorax was rarely found.

INCIDENCE OF HEMOTHORAX IN PRIMARY CARCINOMA OF THE LUNG

In all the cases accepted as primary carcinoma of the lung, the diagnosis was made by autopsy, bronchoscopy, thoracotomy, thoracoscopy, gland biopsy, punch biopsy, or by the finding of carcinoma cells in the pleural fluid. Only after a careful search to rule out all other possible diagnoses was a positive diagnosis of primary carcinoma of the lung accepted. In collecting the instances of primary carcinoma of the lung with hemothorax which are used in this series, many cases in which a diagnosis of primary carcinoma of the lung was made entirely on symptomatology or by x-ray findings were excluded. Of the total of 247 acceptable instances of primary carcinoma of the lung, thirty-five, or 14 per cent of the total, showed bloody pleural fluid. Some patients had primary carcinoma of the lung in the late stage, with pleural involvement, which may account for the rather frequent finding of bloody fluid in the pleural space. The findings in these thirty-five cases of proved carcinoma of the lung with hemothorax are presented in Table

TABLE II

CASE	AGE (YR.)	SEX	PAIN	COUGH	DYSPNEA	BLOODY SPUTUM	FATIGUE	WEIGHT LOSS	SIGNS	LOCATION	TYPE OF CARCINOMA	HOW DIAGNOSIS CONFIRMED	PLEURAL FLUID	BRONCHOSCOPY	X-RAY DIAGNOSIS
1	37	M	+						Fluid	Left base	No type	Autopsy	Bloody; tumor cells		Fluid and tumor, left base
2	45	F	+	+	+	+			Fluid	Right	Adenocarcinoma	Autopsy	Bloody; no cells	Neoplasm pressing on wall of right bronchus	
3	83	F		+	+				Fluid	Right base	Adenocarcinoma	Biopsy; autopsy	Bloody		Fluid right chest, after tap, pathologic process in right base
4	38	M	+	+		+			Fluid	Left	No type	Autopsy	Bloody; no cells		
5	60	M	+	+		+			Dullness	Right apex	Pancoast tumor	Autopsy			Dull right apex and cavity; fluid; malignant process
6	45	F	+		+				Fluid	Left base	Adenocarcinoma	Autopsy	Bloody		Fluid on left
7	42	F		+	+				Fluid	Left base	Adenocarcinoma	Autopsy	Bloody; no cells	Negative	Fluid on left; ? implants right
8	48	M	+						Dullness	Right apex	Epidermoid carcinoma	Autopsy			Mass at right hilus

9	40	F		+		+		+	Dull-ness	Right apex	Adeno-carci-noma	Autopsy			Mottled dullness in upper half right lung field
10	63	M		+	+					Right	Adeno-carci-noma	Autopsy	Bloody; tumor cells		
11	37	M							Dull-ness	Right base	Oat-cell carci-noma	Autopsy	Dry tap		Dull right base; con-solidation
12	65	M		+	+				Dull-ness	Left apex	Epider-moid carci-noma	Autopsy			Tumor in left upper lobe; carcinoma
13	56	M			+				Fluid	Right base	Epider-moid carci-noma	Autopsy			
14	83	F	+		+				Fluid	Right base	Adeno-carci-noma	Biopsy; autopsy	Bloody; no cells		Pathologic process, right base
15	57	M	+							Right	Oat-cell carci-noma	Autopsy	Bloody		Neoplasm right pleura
16	33	F	+						Fluid	Left	Adeno-carci-noma	Autopsy	Bloody fluid at apex		Bronchial stenosis and carcinoma
17	62	F	+	+					Fluid	Right	Adeno-carci-noma	Bron-chos-copy	Bloody fluid at apex	Nodule in right main bronchus opposite orifice of middle lobe; Biopsy: adenocarcinoma	Fluid and atelectasis

TABLE II (CONT'D)

CASE	AGE (YR.)	SEX	PAIN	COUGH	DYSPNEA	BLOODY SPUTUM	FATIGUE	WEIGHT LOSS	SIGNS	LOCATION	TYPE OF CARCINOMA	HOW DIAGNOSIS CONFIRMED	PLEURAL FLUID	BRONCHOSCOPY	X-RAY DIAGNOSIS
18	53	M		+	+	+			Fluid	Left	Oat-cell carcinoma	Bronchoscopy	Bloody; no cells	Bleeding mass in left main bronchus; involvement outside bronchi with secondary invasion; Biopsy: oat-cell carcinoma	Fluid when removed air was replaced; left hilar mass; carcinoma
19	54	M				+	+	+	Fluid	Left	No type	Thoracotomy	Bloody; no cells	Compression of left lower lobe; hemorrhagic lesion; ? malignancy; Biopsy: chronic inflammatory tissue	Fluid, density suggesting parenchymal lesion
20	52	M	+						Fluid	Right	Adenocarcinoma	Thoracotomy	Bloody	Trachea encroached upon from right side above carina; nothing in bronchial tree	Effusion; air replaced; mass in right hilus; ? malignancy
21	53	F	+		+				Dullness	Right	No type	Thoracoplasty	Bloody; tumor cells		Fluid right; infiltrative process in lower and middle lobes
22	31	M	+	+					Dullness	Left	Endothelioma	Thoracoplasty	Bloody; tumor cells		
23	34	F	+	+					Fluid	Right base	No type	Thoracoplasty	Bloody; no cells	Mass in right lower lobe bronchus	Fluid at right base

24	43	M	+		+				Fluid	Right	Adeno-carci-noma	Thoraco-plasty	Bloody fluid at apex		Fluid and air; pleural masses; lung nodule on right
25	48	M			+				Fluid	Left	No type	Thoraco-plasty	Bloody; no cells		Fluid air replaced; pleural malignancy
26	50	M					+		Fluid	Right	Adeno-carci-noma	Biopsy	Bloody; no cells		Dull right chest
27	52	M	+	+	+					Left	Adeno-carci-noma	Biopsy	Bloody		
28	47	M	+			+			Fluid	Right	No type	Gland biopsy	Bloody; no cells		Mass right hilus new growth
29	35	M							Fluid	Right	No type	Biopsy	Bloody		Hydropneumothorax
30	50	M	+	+	+				Fluid	Left	Adeno-carci-noma	Gland biopsy; pleural fluid	Bloody; tumor cells		Fluid left; ? meta-static malignancy in right lung
31	32	F	+						Fluid	Left	No type	Gland biopsy	Bloody; no cells		Fluid left
32	30	M			+				Fluid	Left	No type	Punch biopsy	Bloody; no cells	No tumor seen	Fluid left; mass in hilus, carcinoma
33	44	M	+						Dull-ness	Right	Adeno-carci-noma	Pleural fluid	Bloody fluid at apex		Tumor of ribs and pleura
34	55	M		+		+			Fluid	Right	No type	Pleural fluid	Bloody; tumor cells		Fluid and dullness on right
35	69	M	+	+					Fluid	Left	No type	Pleural fluid	Bloody; tumor cells		Primary neoplasm of lung, with fluid

II. In these selected cases, the clinical diagnosis of primary carcinoma of the lung was confirmed by autopsy in sixteen cases, by bronchoscopy in two, by exploratory thoracotomy in two, by thoracoscopy in five, by metastatic gland biopsy in six, by punch biopsy in one, and by tumor cells in the pleural fluid in eleven.

AGE, SYMPTOMS, AND SIGNS

Primary carcinoma of the lung with hemothorax occurred in the latter age decades, as was to be expected from the age incidence of carcinoma in general. The youngest of these thirty-five patients was 30 years old; the oldest was 83. Twenty-three of the thirty-five patients were between the ages of 40 and 65.

Eleven patients, or almost 33 per cent in this series of thirty-five, were women. This is a somewhat higher percentage in this small series than the 20 per cent incidence of primary carcinoma of the lung, with and without hemothorax, in females found in larger series.

The most common symptoms were, in the order of frequency: pain, cough, dyspnea, sputum (bloody in six of eight instances), fatigue, and weight loss. The latter two symptoms occurred, however, in only two patients.

The physical signs elicited upon examination of the chest were mainly those of pleural fluid.

THE RELATION OF HEMOTHORAX TO SITE OF LUNG LESION

In nineteen patients the lesion was in the right lung; in fifteen, in the left lung; and in one instance, the process was bilateral. The primary malignant process occurred anywhere in the lung, no particular site showing any preponderance. Thus, the location of the primary carcinoma in the lung had no definite relation to the presence of hemothorax.

ROENTGENOGRAMS AND HEMOTHORAX

Of the thirty-five patients with hemothorax, twenty-nine had x-ray examinations. A positive diagnosis of malignancy of the lung was made by the roentgenologist in eighteen cases. This diagnosis was usually made because of the presence of a pulmonary mass and fluid. In four cases, pleural fluid and irregularity of the pleura were the diagnostic features. In the differential diagnosis, lung abscess might simulate the shadow cast by a carcinomatous mass, but it is unusual to have pleural fluid concomitant with a lung abscess unless the abscess is ruptured. A fluid level is then usually present as a result of the escape of both air and fluid. Benign tumors of the lung may simulate malignant masses on the x-ray films but they are rarely accompanied by pleural fluid.

BRONCHOSCOPY AND HEMOTHORAX

Only eight patients in this series of thirty-five were bronchoscoped. The reason for the small number of bronchoscopic examinations may be: (1) Some of the individuals were studied many years ago when this procedure was rarely used. (2) The tumor mass seen by x-ray was located away from the main bronchi and it seemed unlikely that the bronchoscopist could visualize it. In only two of the eight patients were the bronchoscopic examinations entirely negative. In two patients biopsy of a bronchial mass revealed carcinomatous cells, one an oat-cell carcinoma and the other an unidentified type of carcinoma. In three patients biopsy could not be performed; in the first, because the neoplasm was pressing on the bronchial wall from without; in the second, because the trachea was encroached upon extrinsically above the carina; and in the third, because there were mechanical difficulties, although a mass was seen in the bronchus. In one patient a hemorrhagic lesion, pronounced malignant by the bronchoscopist, was biopsied and reported as chronic inflammatory tissue. In only one of the eight cases bronchoscoped were carcinoma cells found in the hemorrhagic pleural fluid. Therefore, bronchoscopy should be done in all patients with hemothorax and a pulmonary shadow by x-ray, even if repeated diagnostic taps have shown no tumor cells.

PLEURAL FLUID EXAMINATION IN HEMOTHORAX

The bloody fluid obtained by aspiration was not examined microscopically in twelve cases. The majority of these fell in that group studied many years ago. In the twenty-three patients in whom the pleural fluid was studied, eleven showed carcinomatous cells and twelve did not. In four of the eleven positive cases, a diagnosis of adenocarcinoma was made since the cell type was obvious; in the other seven, merely cancer cells were noted and no definite type could be identified.

Accordingly, almost one-half of the bloody pleural fluids studied showed carcinomatous cells. Schlesinger[2] has found that in 50 per cent or more of all pleural fluids with carcinoma in the lung, whether or not the fluids were bloody, recognizable neoplastic elements were present. With the improved technique as described in his paper, and the establishment of fairly definite criteria upon which to base the diagnosis of malignant cells, the percentage of positive findings upon microscopic examination of bloody pleural fluids should increase. Repeated aspirations and successive microscopic sections are sometimes needed to find tumor cells.

TYPE OF PRIMARY CARCINOMA OF THE LUNG WITH HEMOTHORAX

Fifteen of the thirty-five patients studied had adenocarcinoma, twelve had carcinoma without classification, three had epidermoid carcinoma,

three had oat-cell carcinoma, one had an endothelioma, and one a Pancoast tumor.

Primary adenocarcinoma of the lung, occurring in 45 per cent of the patients studied, was therefore the predominant type associated with hemothorax. Olson[3] and Arkin and Wagner[4] found that the incidence of adenocarcinoma was 25 per cent of all primary carcinomas of the lung.

METASTATIC PULMONARY NEOPLASM WITH HEMOTHORAX

Hemothorax in metastatic pulmonary neoplasm occurred only twice, the primary lesions being in the breast and pancreas, respectively. It is therefore uncommon to have bloody pleural fluid and secondary pulmonary malignant metastases coexist. Thus, when carcinomatous cells are found in a pleural fluid which is bloody, with or without a definite shadow by x-ray, the primary lesion will almost always be in the pulmonary parenchyma.

SIGNIFICANCE OF HEMOTHORAX IN PRIMARY CARCINOMA OF THE LUNG

Surgical intervention is contraindicated (1) in those instances of hemothorax in which carcinomatous cells are found, and (2) in those cases of hemothorax showing no tumor cells by aspiration, but where malignancy is demonstrated by bronchoscopy. In all such patients explored, the lesion was found to have spread beyond the hope of surgical control or extirpation.

SUMMARY

The etiology of eighty-six cases of hemothorax is discussed.

The incidence of hemothorax in 247 proved cases of primary carcinoma of the lung is determined.

An analysis is made of the age, symptoms, signs, x-rays, and site of the lung lesion in these patients with carcinoma of the lung and hemothorax.

The value of bronchoscopy in hemothorax without cancer cells is suggested.

The type of primary carcinoma of the lung with hemothorax is tabulated, and comment is made upon the rather uncommon association of metastatic pulmonary neoplasm with hemothorax.

The significance of hemothorax in primary carcinoma of the lung is shown.

I wish to acknowledge the kindness of Dr. Churchill, Dr. Overholt, and Dr. Strieder in permitting me to review their cases.

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The American Association for Thoracic Surgery
Twenty-Fourth Annual Meeting
Toronto, Ontario, Canada
June 9, 10, and 11, 1941

Program

MONDAY, JUNE 9

9:00 A.M. Business session.

9:30 A.M. Scientific session

1. Anatomic Change in the Lungs Following Thoracoplasty
   Oscar Auerbach (by invitation), Staten Island, N. Y.
2. Surgery in Pulmonary Tuberculosis.
   Ralph Adams and (by invitation) Paul DuFault, Boston, Mass.
3. Thoracoplasty for Pulmonary Tuberculosis.
   George F. Skinner, St. John, New Brunswick.
4. Thoracoplasty for Pulmonary Tuberculosis.
   1. Analysis of One Hundred Consecutive Cases of Thoracoplasty With No Mortality.
      Richard H. Dieffenbach and (by invitation) Anthony D. Crecca, Newark, N. J.
   2. Analysis of 104 Cases of Thoracoplasty for Pulmonary Tuberculosis.
      George G. Finney, Baltimore, Md.
   3. The Results of Thoracoplasty.
      H. Meltzer, Ninette, Manitoba.
   4. Thoracoplasty in Bilateral Pulmonary Tuberculosis.
      Arthur M. Vineberg and (by invitation) Douglas Ackman and Michael Aronovitch, Montreal, Quebec.

5. Results in 90 Consecutive Thoracoplasties for Pulmonary Tuberculosis.
Arthur H. Aufses (by invitation), New York, N. Y.

5. Extra-Periosteal Pneumonolysis in Pulmonary Tuberculosis.
Charles P. Bailey (by invitation), Philadelphia, Pa.

2:00 P.M. Scientific session

6. Bronchospirography. Studies of the Functional Capacity of Re-expanded Lungs Following Pneumothorax and of Lungs Following Thoracoplasty.
Max Pinner and (by invitation) George Leiner and William A. Zavod, New York, N. Y.

7. Studies of Individual Lung Function.
Walter Whitehead (by invitation), Detroit, Mich.

8. Bronchospirography.
George W. Wright (by invitation), Trudeau, N. Y., and Warriner Woodruff, Saranac Lake, N. Y.

9. A Review of 100 Consecutive Cases of Endobronchial Tuberculosis.
J. Maxwell Chamberlain, Oneonta, N. Y.

10. Tuberculous Stenosis of the Major Bronchi.
William M. Tuttle, Detroit, Mich.

8:30 P.M. Scientific session

11. Effect of Thoracoplasty Upon Pulmonary Tuberculosis Complicated by Stenotic Tuberculous Bronchitis.
John Alexander, Ann Arbor, Mich., George N. J. Sommer, Jr., Trenton, N. J., and Adrian A. Ehler, Albany, N. Y.

12. A Preliminary Report on Some Investigative Procedures Regarding Closure of Refractory and Residual Cavities.
Louis R. Davidson, New York, N. Y.

13. Open Drainage for Residual Cavities Beneath Thoracoplasty.
W. L. Rogers, Sidney J. Shipman, and (by invitation) A. C. Daniels, San Francisco, Calif.

TUESDAY, JUNE 10

9:00 A.M. Scientific session

14. Surgical Significance of Metastasis in Primary Carcinoma of Lung: Report of Two Cases With Unusual Site of Metastasis.
Alton Ochsner and Michael DeBakey, New Orleans, La.

15. A Topographic Classification of Cancer of the Lung With Special Reference to the Surgical Implications of the Circumscribed Variety.
Harold Neuhof and (by invitation) Coleman B. Rabin and Irving A. Sarot, New York, N. Y.

16. Pneumonectomy for Carcinoma of the Lung.
Stuart W. Harrington, Rochester, Minn.

17. Experiences in Total Pneumonectomy.
Norman S. Shenstone (by invitation), Toronto, Ontario.

18. Pulmonary Function After Pneumonectomy in Children.
Charles W. Lester, and (by invitation) André Cournand and Robert L. Riley, New York, N. Y.

2:00 P.M. Executive session.

3:00 P.M. Presidential Address. Pulmonary Abscess.

Fraser B. Gurd, Montreal, Quebec.

19. Pulmonary Abscess.
Robert R. Shaw, Dallas, Texas.

20. Chronic Non-Tuberculous Thoracic Empyema.
Albert O. Singleton and (by invitation) Robert M. Moore, Galveston, Texas.

21. Surgery of the Chest in War.
A. L. Lockwood, Toronto, Ontario.

7:30 P.M. Annual Dinner at the Royal York Hotel.

Address: Aviation Medicine.
Wing Commander G. E. Hall, R.C.A.F.

WEDNESDAY, JUNE 11

9:00 A.M. Scientific session

22. A Method of Treatment of Large Pulmonary Air Cysts (Balloon Cysts) by an Endocutaneous Flap.
A. Lincoln Brown and William Brock (by invitation), San Francisco, Calif.

23. Cancer of the Thoracic Esophagus and the Upper End of the Stomach: A Review of Nineteen Cases That Have Undergone Surgical Exploration.
H. Brodie Stephens, San Francisco, Calif.

24. Experiences With Eight Cases of Resection for Carcinoma of the Esophagus.
Dallas B. Phemister (by invitation), Chicago, Ill.

25. Carcinoma of Esophagus—Demonstration of Cases.
Harold W. Wookey (by invitation), Toronto, Ontario.

26. An Experimental Study of Tubes Made From the Greater Curvature of the Stomach.
B. Noland Carter and (by invitation) Osler A. Abbott and C. Rollins Hanlon, Cincinnati, Ohio.

2:00 P.M. Scientific session

27. Atelectasis and Bronchiectasis.
Joseph Tannenberg (by invitation) and Max Pinner, New York, N. Y.

28. The Use of Sulfanilamide in Resection of the Lung.
Frank Allbritten (by invitation), John B. Flick, and John H. Gibbon, Jr., Philadelphia, Pa.

29. Experimental Study of the Effect on Cardiorespiratory Function of Blood Transfusion Following Lobectomy.
John H. Gibbon, Jr., Philadelphia, Pa.

30. Major Changes in the Fundamental Relationships of the Respiratory Drive Mechanisms During Evipal and Pentothal Anesthesia, With Special Consideration of Possible Applications to Transpleural Surgery.
Carl A. Moyer (by invitation), Boston, Mass.

31. Observations on the Effects of the Prolonged Administration of High Oxygen Concentrations to Dogs.
John R. Paine (by invitation), Minneapolis, Minn.